

WHO OGCT AS A DIAGNOSTIC TEST FOR GESTATIONAL DIABETES MELLITUS

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**INSTITUTE OF OBSTETRICS AND GYNAECOLOGY
MADRAS MEDICAL COLLEGE, CHENNAI-3.
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CERTIFICATE

This is to certify that the dissertation entitled “**WHO OGCT AS A DIAGNOSTIC TEST FOR GESTATIONAL DIABETES MELLITUS**” is the bonafide original work of **Dr. D. PREETHI** in partial fulfilment of the requirements for **M.D. Branch – II (Obstetrics and Gynaecology)** Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in March 2007.

Prof. Dr. ANJALAKSHI CHANDRASEKAR,
M.D. , D.G.O., Ph.D.
Professor, Department of Obstet & Gynaec.
Institute of Obstetrics and Gynaecology
Madras Medical College
Chennai-600 003.

Director and Superintendent
Institute of Obstetrics and Gynaecology
Madras Medical College
Chennai-600 003.

Dr. KALAVATHY PONNIRAIVAN, B.Sc., M.D.
DEAN
Madras Medical College & Hospital,
Chennai-600 003.

DECLARATION

I, **Dr. D. PREETHI**, solemnly declare that dissertation titled, “**WHO OGCT AS A DIAGNOSTIC TEST FOR GESTATIONAL DIABETES MELLITUS**” is a bonafide work done by me at Institute of Obstetrics and Gynaecology, Madras Medical College & Hospital during 2005-2006 under the guidance and supervision of **Prof. Dr. ANJALAKSHI CHANDRASEKAR, M.D. , D.G.O., Ph.D.**

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(Dr. D PREETHI)

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ABBREVIATIONS

ADA	:	American Diabetes Association
AGT	:	Abnormal Glucose Tolerance
BMI	:	Body Mass Index
BW	:	Birth Weight
DM	:	Diabetes Mellitus
EDTA	:	Ethylene Diamine Tetra Acetic Acid
FPG	:	Fasting Plasma Glucose
G.A	:	Gestational Age
GDM	:	Gestational Diabetes Mellitus
GOD	:	Glucose Oxidase
GTT	:	Glucose Tolerance Test
HAPO	:	Hyperglycemia Adverse Pregnancy Outcome
Hb	:	Hemoglobin
IGT	:	Impaired Glucose Tolerance
IOG	:	Institute Of Obstetrics And Gynecology
IQ	:	Intelligent Quotient
IUD	:	Intra Uterine Death
NDDG	:	National Diabetes Data Group
NonGDM	:	Non Gestational Diabetes Mellitus
OGCT	:	Oral Glucose Challenge Test
OGTT	:	Oral Glucose Tolerance Test
PCV	:	Packed Cell Volume
PIH	:	Pregnancy Induced Hypertension
POD	:	Peroxidase
PPG	:	Post Plasma Glucose
RDS	:	Respiratory Distress Syndrome
SD	:	Standard Deviation
SE Status	:	Socio Economic Status
UTI	:	Urinary Tract Infection
VSD	:	Ventricular Septal Defect
WHO	:	World Health Organisation

KEY TO MASTER CHART

Age

17-20 years	-	1
21-25 years	-	2
26-30 years	-	3
31-35 years	-	4
36 - 40 years	-	5

Socio Economic Status

Class I	-	A
Class II	-	B
Class III	-	C
Class IV	-	D
Class V	-	E

BMI

≤ 20	-	A
21– 24	-	B
25 - 29	-	C
≥ 30	-	D

FAMILY HISTORY

Positive	-	+
Negative	-	-

GESTATIONAL AGE AT TESTING

16-20 weeks - 1
21-24 weeks - 2
25-28 weeks - 3
29-32 weeks - 4

OBSTETRICS CODE

G1(Gravida 1) - I
G2 (Gravida 2) - II
G3(Gravida 3) - III
G4 (Gravida 4) - IV
G5 (Gravida 5) - V
G6 (Gravida 6) - VI

RISK FACTORS IN PREVIOUS PREGNANCY

Spontaneous Abortion	-	SA
Macrosomia	-	M
Sudden Intrauterine Death	-	SID
Unexplained Still Birth	-	USB
Unexplained Neonatal Death	-	UND
History of GDM	-	HG
History of PIH	-	HP
Congenital Anomaly	-	CA

RISK FACTORS IN PRESENT PREGNANCY -

Obesity	-	OB
Pregnancy Induced Hypertension	-	PIH
Hydramnios	-	H

Glucosuria	-	G
Congenital Anomaly	-	CA
Macrosomia	-	M
Recurrent UTI	-	RU
Moniliasis	-	MON

GESTATIONAL AGE AT DELIVERY

Term	-	T
Preterm	-	PT

BIRTH WEIGHT (Kg)

2 – 2.4	-	A
2.5 – 2.9	-	B
3 – 3.4	-	C
3.5 – 3.9	-	D
≥ 4	-	E

APGAR SCORE

8 and above	-	A
5 – 7	-	B
1 – 4	-	C

NEONATAL COMPLICATIONS

Hypoglycemia	-	HG
Hypocalcemia	-	HC
Hyperbilirubinemia	-	HB
Respiratory Distress Syndrome	-	RDS
CA	-	Congenital Anomaly

INTRODUCTION

Diabetes Mellitus is a clinical syndrome characterized by hyperglycemia due to absolute or relative deficiency of insulin. Lack of insulin whether absolute or relative affects the metabolism of carbohydrate, protein, fat⁽¹⁾.

Pregnancy is characterised by mild fasting hypoglycemia, post prandial hyperglycemia, hyper insulinism and insulin resistance- a *diabetogenic stress*. Normally pregnant woman elaborates an increased insulin production by 30% above her non pregnant state. A woman who is unable to achieve adequate insulinogenic compensation develops Gestational Diabetes. Pregnancy unmasks the minor intolerance of carbohydrate metabolism in subjects with reduced pancreatic islet cell reserve.⁽²⁾

Gestational Diabetes was defined as carbohydrate intolerance of variable severity with its onset or first recognition during pregnancy. Use of this term was encouraged in order to communicate the need for increased surveillance and to convince the woman of the need for further testing postpartum.⁽³⁾

Gestational Diabetes is often asymptomatic and associated with increased fetal and neonatal morbidity and mortality. Good glycemic control reduces the risk of complication⁽⁴⁾.

CARBOHYDRATE METABOLISM IN NON DIABETIC PREGNANCY(5)

Factors contributing to insulin resistance

Production of placental somatomammotrophin, increased production of estrogen, progesterone, increased insulin destruction by placental enzyme like insulinase.

Changes in Gluconeogenesis

Fetus continuously uses fuels from the mother. It uses alanine and other amino acids and depletes the mother of a major gluconeogenic source.

Increased Lipolysis

The mother uses fat for caloric needs and saves glucose for the fetus.

METABOLIC CHANGES DURING FASTING

During fasting there is decrease in plasma glucose and amino acids. There is higher plasma concentration of free fatty acids, triglycerides. During fasting for a longer period there is switch in metabolism from glucose to lipid which is termed “*accelerated starvation*” by Freinkel⁽⁸⁾

METABOLIC CHANGES DURING FED STATE⁽⁵⁾

During the first few hours, glucose absorbed from the gastro intestinal tract provides for the metabolic needs of brain and other organs. The absorbed glucose in excess of these needs is used to rebuild fuels in liver, muscle , fat and to provide supply of glucose for the fetus. This is *facilitated anabolism*.

REVIEW OF LITERATURE

HISTORY

Diabetes was described more than 2000 years ago. An ancient documentation by *Susruta* in India at about 400 B.C. has described the diabetic syndrome as characterized by a “honeyed urine”. The word Diabetes (to flow through) was coined by Greek physician *Aretaus* of Cappadocia in first century (150 A.D) from the word siphon (sweet taste). The word mellitus (honeyed) was added by John Rollo in 18th century. In 1674, *Thomas Willis*, a Physician, Anatomist and a professor of Natural philosophy at Oxford discovered by tasting that the urine of diabetic persons was “wonderfully sweet as if imbued with honey or sugar”. Willis could not explain the chemical nature of the sweet substance. It was *Mathew Dobson* of Manchester, England who in 1776 demonstrated that diabetics actually excrete sugar in urine. It was John Rollo, surgeon general of Royal artillery who first applied the discovery of glycosuria by Dobson to the quantitative metabolic study of diabetes.

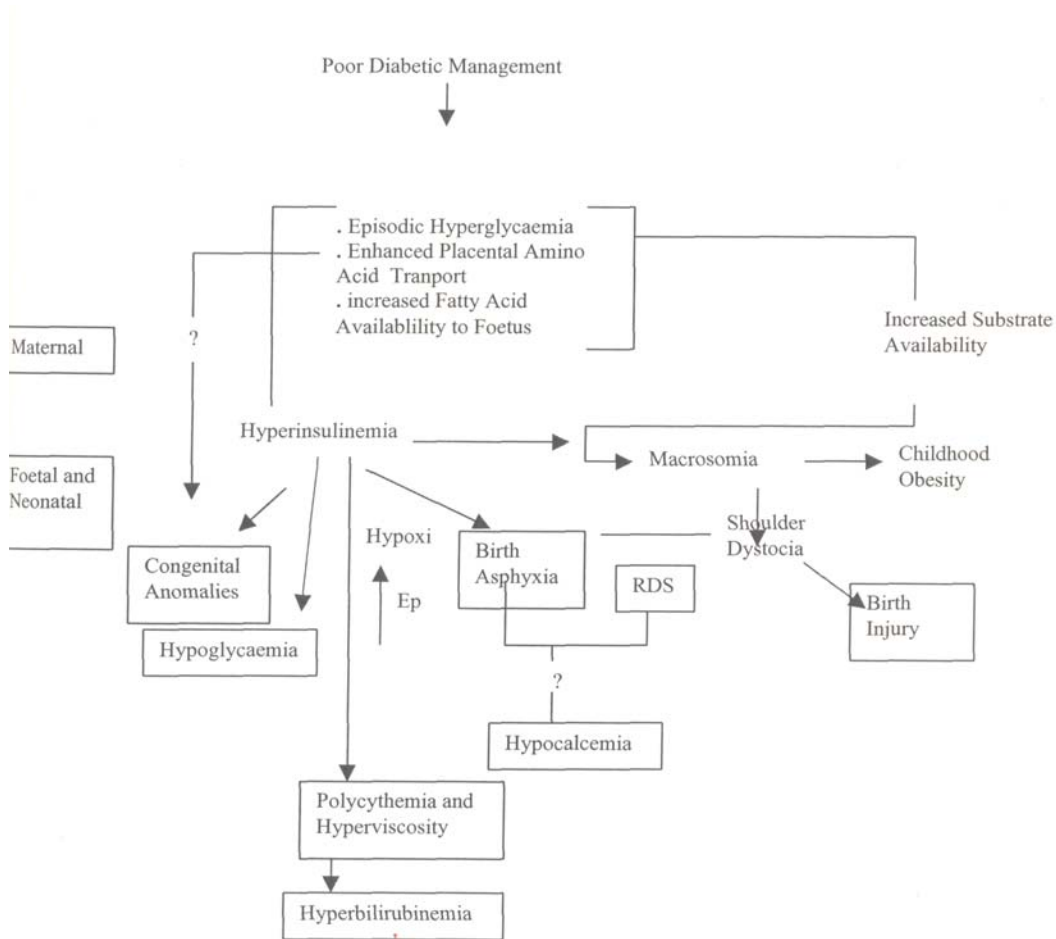
It was *Claude Bernard* who studied the association between pancreas and Diabetes. The name Insulin was coined by *De Mayer* (1909). In 1921 *Fredrick Banting* and *Charles Best* with the help of Chemist *J.B.Collip* succeeded in fulfilling all of the criteria for therapeutic active Insulin.

Glucose homeostasis reflects a precise balance between hepatic glucose production and peripheral glucose uptake and utilisation. Insulin is the most important regulator of this metabolic equilibrium, but neural input, metabolic signals and hormones result in integrated control of glucose supply and utilization. In the fasting state low insulin levels increase glucose production by promoting hepatic gluconeogenesis and glycogenolysis and reduce glucose uptake in insulin sensitive tissue. Glucagon also stimulates glycogenolysis and gluconeogenesis by the liver and renal medulla. Post prandially the glucose load elicits a rise in insulin and fall in glucagon. The major portion of post prandial glucose is utilized by skeletal muscles, an effect of insulin stimulated glucose uptake⁽⁶⁾.

PATHOPHYSIOLOGY OF PERINATAL MORBIDITY

The Pedersen hypothesis suggested that in diabetic pregnancy maternal hyperglycaemia is rapidly translated into foetal hyperglycaemia. The foetal pancreas responds to this glycaemic stimulus with islet cell hypertrophy and hyperplasia and foetal hyperinsulinism results. It is the foetal hyperinsulinemia that results in diabetic fetopathy or perinatal morbidity seen in such pregnancies. According to modified Pedersen hypothesis the non glucose secretagogues for foetal pancreatic insulin also play a role in perinatal morbidity⁽⁵⁹⁾.

PATHOGENESIS OF PERINATAL MORTALITY AND MORBIDITY

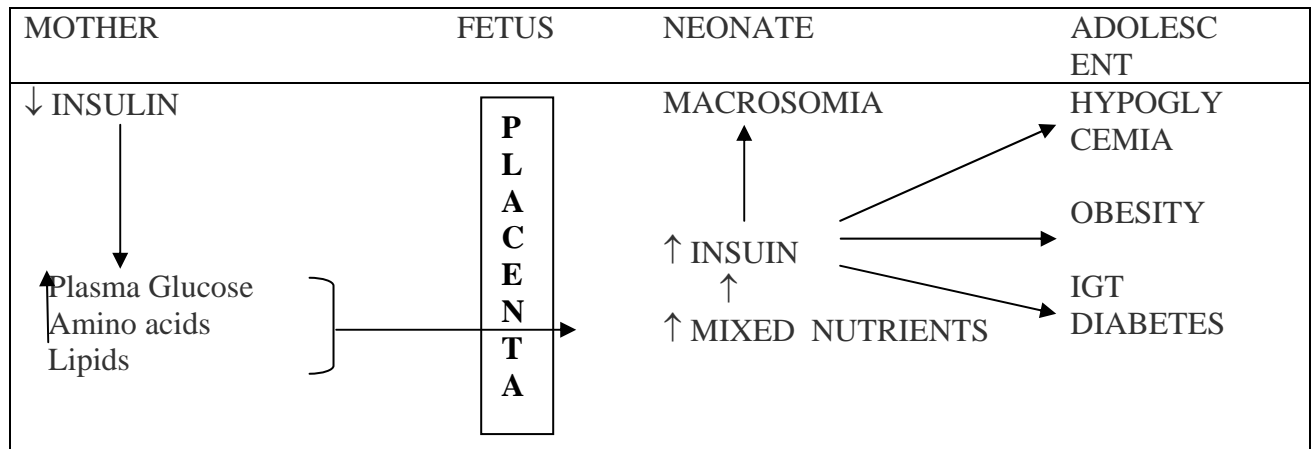


MODIFIED PEDERSEN / FRIENKEL'S HYPOTHESIS

The transport of maternal fuel to the foetus requires normal placental intermediary metabolism and normal supply of substrates. Because diabetes may result in markedly abnormal concentrations of maternal glucose, fatty

acids, triglycerides and amino acids these may get transported to the foetus.

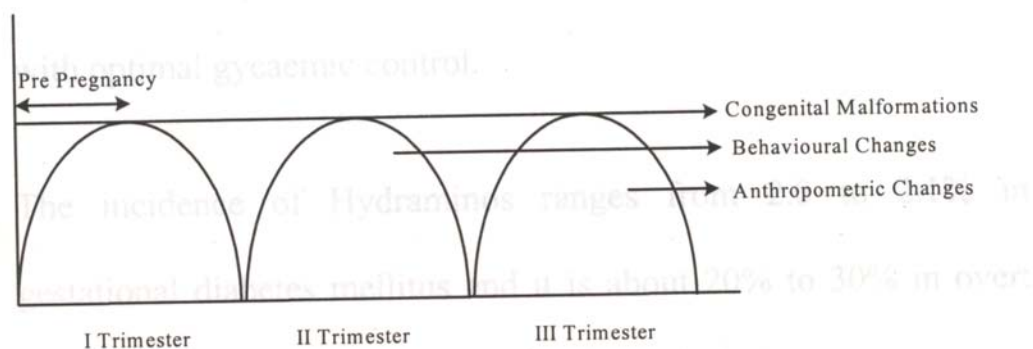
Unlike the foetus of early gestation, the foetus of the late gestation is well equipped to synthesise and replace insulin from its pancreas and protect itself against brunt of abnormal fuel mixture, thereby normalizing blood sugar in circulation but in the process resulting in hyperinsulinemia of the foetus and due to its anabolic action causes macrosomia and associated complications.



Gestational carbohydrate intolerance is asymptomatic. The subjects destined to develop gestational diabetes mellitus have limited pancreatic insulin reserve and reduced insulin sensitivity. The stress of pregnancy due to counter insulin hormones overwhelms the insulin reserves. Hence in the plasma, levels of all classes of fuels – aminoacids, fatty acids and glucose are elevated which are delivered to the foetus. This change is seen more during the latter half of pregnancy when counter insulin factors and insulin

resistance is experienced. The foetal transportation of abnormal fuels result in hyperinsulinemia and the resultant macrosomia. Thus macrosomia and associated risk factors like traumatic delivery, RDS, neonatal hypoglycemia, hypocalcaemia, hyperbilirubinemia become the major concern for the subjects with gestational diabetes.

FRIENKEL'S FUEL MEDIATED TERATOGENESIS



Effects of abnormal glucose tolerance on the mother and the foetus:

On the mother:

- Pre-eclampsia and pregnancy induced hypertension⁽⁴⁰⁾ is seen in 13.7% in Gestational Diabetes Mellitus, and 14.1% to 27% in established diabetes mellitus. Pre - eclampsia and pregnancy induced hypertension are more common in patients with Gestational Diabetes than in controls⁽⁴¹⁾. ***Combs et***

al and Rosenn et al⁽⁴²⁾ reported a significant association between poor glycaemic control and pre-eclampsia or pregnancy induced hypertension.

- The incidence of chronic hypertension⁽⁴³⁾ is 2.5% among Gestational Diabetes, against 0.3% in the non diabetic control group.
- Overall incidence of ketoacidosis is 0.7% especially following beta agonist therapy. *Kilvert et. al.*, reported one case of diabetic ketoacidosis in 150 cases of Gestational Diabetes Mellitus.⁽⁴⁴⁾ Diabetic ketoacidosis is preventable and the prevention can be accomplished with optimal glycemic control.
- The incidence of Hydramnios ranges from 2.0 to 2.1%⁽⁴⁵⁾ in gestational diabetes mellitus and it is about 20% to 30% in overt diabetics. Most infants of hydamniotic diabetic pregnancy are structurally normal, associated with increased incidence of preterm labour and premature rupture of membrane.
- Pyelonephritis was reported in 1.2% of gestational diabetes mellitus and 3.6% of overt diabetes⁽⁴³⁾. There is no difference in the incidence of pyelonephritis in gestational diabetes mellitus and control groups.
- Preterm labour complicated 8.1% of gestational diabetes mellitus and there is no significant difference in the preterm labour rate between gestational

diabetes mellitus and control groups. There is significant correlation between preterm labour and uro-genital infection. (Candida and Trichomoniasis). **Molsted and Pedersen**⁽⁴⁶⁾ speculated that, hormonal differences increased the frequency of preterm labour in a diabetic than in non-diabetic women.

- Spontaneous pre-term delivery is one of the important contributors to perinatal mortality in diabetic pregnancies⁽⁴⁶⁾. In a Scandinavian report by **Molsted and Pedersen**, the incidence of preterm labour with delivery was 14.6% in gestational diabetes mellitus versus 18 to 24% in established diabetes mellitus, and in the control group it was 12%.
- The incidence of primary caesarean section among gestational diabetes mellitus women ranged from 13.4% to 18.4%⁽⁴⁷⁾ and in controls 12.9% but repeat section in gestational diabetes mellitus was 16.5% and 6% among the control groups. There is a higher total caesarean section rate in gestational diabetes mellitus than controls⁽⁵⁵⁾.

Foetal Problems associated with maternal hyperglycemia

First Trimester	Second trimester	Third Trimester
Malformations	Hypertrophic Cardiomyopathy	Hypoglycemia
Growth retardation	Polyhydramnios	Hypocalcemia
Foetal wastage	Placental insufficiency	Hyperbilirubinemia

	Pre-eclampsia Foetal loss Low IQ	Respiratory – Distress Syndrome Macrosomia Hypomagnesemia Intrauterine death
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- Hypoglycemia is one of the common causes for perinatal morbidity. It is defined as blood sugar level less than 40mg% in any infant regardless of gestational age. About 50% of the hypoglycemic babies may remain asymptomatic. The factor mainly protective against foetal hypoglycemia is the optimal control of maternal hyperglycemia especially during the third trimester and during labor. It has been shown that a mean maternal plasma glucose >105 mg/dl during the last four hours of labour in a diabetic mother leads to a higher incidence of neonatal hypoglycemia.
- About 25% of the infant of diabetic mothers may present with serum calcium of <7 mg/dl and this may remain mostly asymptomatic and is usually detectable during the 2nd and 3rd day of birth. Hypomagnesemia may coexist and may require correction.
- Respiratory distress syndrome (RDS) occurs in about 5% of the infant of diabetic mothers and it is seen equally in gestational diabetes mellitus. Again a strict glycaemic control reduces the incidence of RDS.
- Polycythemia is relatively common in infant of diabetic mother. The hyperviscosity due to Polycythemia may induce congestive heart failure and

vascular thrombosis accounting for the increased risk of renal vein thrombosis in these infants.

- Hyperbilirubinemia, the common abnormality is due to increased bilirubin production and decreased life span of the RBCs with glycosylated cell membranes. Hepatic conjugation of bilirubin may be impaired due to an immature liver.

Gestational Diabetes Mellitus is mostly the forerunner of Type-2 Diabetes Mellitus⁽⁹⁾. Like type-2 Diabetes mellitus ,obesity and advanced maternal age increase the risk of gestational diabetes mellitus^(10,11).

PREVALENCE

Ethnically Indian women are more prone to develop glucose intolerance during pregnancy and have eleven fold increased risk compared to white caucasian necessitating universal screening during pregnancy⁽⁷⁾.

Prevalence of Gestational Diabetes is 2% to 5% of all pregnancies in the UnitedStates⁽¹²⁾. Marked variation has been reported in the prevalence of gestational diabetes world wide. The frequency of gestational diabetes mellitus ranged widely from 0.15% to as high as 50% in pima Indians in the USA.⁽¹³⁾.

Ethnic and Racial differences in prevalence

Hispanic, Native American, Asian, African American are racial / ethnic groups with an increased risk of developing diabetes. Asian races are pointed out

as one of the groups with highest prevalence of diabetes ⁽¹⁴⁾. Like racial and ethnic groups, positive family history of diabetes, older mothers, heavier women are the other risk factors. Woman of oriental, first generation Hispanics from Indian subcontinent and middle east are at an increased risk for Gestational Diabetes ⁽¹⁵⁾. According to Beischer et al 1991, the prevalence of GDM in Indian subcontinent is 15%.

Screening for GDM is important as detection and treatment of GDM is helpful in reducing maternal and perinatal mortality and morbidity.

Following are the risk factors for screening of GDM.

- a) Age > 25 years
- b) Diabetes Mellitus in first degree relatives.
- c) Multiparity
- d) Previous GDM
- e) Past history of large for dates baby
- f) Past history of still birth
- g) Repeated pregnancy loss
- h) Previous congenital malformations
- i) Past history of difficult labor
- j) Recurrent vaginal moniliasis
- k) Recurrent UTI
- l) Maternal obesity

- m) Hydramnios
- n) Pregnancy induced hypertension
- o) Glucosuria
- p) Previous history of unexplained neonatal death

SCREENING TEST :

Various Screening Tests for GDM are

- 1) Urine glucose (Glucosuria)
- 2) Spot Test⁽⁷⁾
- 3) Fasting Plasma Glucose
- 4) Random Plasma Glucose
- 5) O.Sullivan's Oral Glucose Challenge Test
- 6) WHO OGCT
- 7) Glycosylated Hemoglobin (HbA1c)
- 8) Fructosamine

1) URINE GLUCOSE (GLUCOSURIA)

Glucosuria occurs in pregnancy due to the fact that there is increased glomerular filtration rate, intermittent tubular defect in glucose absorption and reduced renal threshold⁽⁷⁾. In a study by Sutherland et al it was found that 11% of obstetric population had glucosuria at sometime but fewer than 1% with glucosuria had abnormal GTT. Detection of glucose in the urine is the simplest

screening procedure. Unfortunately glucosuria is less specific as a screening test⁽¹⁶⁾.

2) SPOT TEST⁽⁷⁾

Seshiah et al in 1984, used spot glucose test to detect glucose intolerance during pregnancy as an alternative simple screening procedure. 971 pregnant women at various stages of gestation at Institute of Obstetrics and Gynecology, Chennai were screened with spot test. Venous blood was obtained at the time of consultation after ascertaining the time at which the subject last had anything to eat. Interval between the time of last meal and time of collection of blood sample was corrected to the nearest 30 minutes. Interval of over 3 hours was interpreted as fasting sample. Whole blood glucose was estimated by O-Toluidine method. The +2 SD figure of the spot test blood glucose is 85mg% fasting and 105mg% non fasting. The approximate plasma glucose values will be around 90mg% and 120mg% respectively. In a pregnant women the fasting and the non fasting glucose value never exceed the above figures. This test has value of sensitivity and specificity comparable to O-Sullivan's screening test.

3) FASTING PLASMA GLUCOSE

Sacks et al ⁽¹⁷⁾ and *Daniele et al* ⁽¹⁸⁾ observed that fasting plasma glucose is an easier screening procedure and suggested a cut off value of 95mg% for GDM. However such level is insufficient as the sole marker of GDM since most cases have FPG values below the putative threshold⁽¹⁹⁾. Very few women are

diagnosed with GDM on the basis of elevated fasting plasma glucose. Nowadays fasting plasma glucose is not considered as a screening test.

4) RANDOM PLASMA GLUCOSE

Random blood glucose is a simple way to screen for abnormal glucose tolerance. A positive cut off was taken as 115mg%, if women were tested less than 2 hrs after a meal and 105mg% if more than 2 hr after a meal. It was 40% sensitive and 90% specific⁽¹⁶⁾. According to *Stangenberg et al* the threshold value for Random blood glucose was 116mg%. With this cutoff, only 11.6% showed abnormal value. Random plasma glucose is useful for a noncompliant patient or in circumstances in which glucose challenge drinks are not available.

5) O.SULLIVAN'S ORAL GLUCOSE CHALLENGE TEST.

It is otherwise known as universal screening test of O Sullivan. This consists of giving a 50gm glucose irrespective of the patients feeding state and time of day and estimating plasma glucose after 1 hour and taking cut off of $\geq 140\text{mg}/100\text{ml}$ for performing the diagnostic 100gm 3 hour oral glucose tolerance test. It was found that when the cut off of 140mg/dl was used it would identify 80% of women with Gestational Diabetes. Using a value of 130mg/dl or more increases the yield to more than 90%⁽⁷⁾. This procedure involves the patient waiting for 1 hour following glucose ingestion and positive screening test to have a second glucose load for diagnosis and it is costly.

6) WHO OGCT

Like WHO OGTT WHO suggested an oral glucose challenge test with same glucose load (75g) and estimation of glycemia after 2 hr for screening. WHO detected an intermediate state between diabetes mellitus and normal glucose tolerance, the reduced glucose tolerance. Normal plasma glucose by this oral glucose challenge test is < 140mg% in 2 hours after 75g glucose load³³. The cut off for diabetes mellitus is > 200mg% and that of impaired glucose tolerance is between 140-200. The impaired glucose tolerance is GDM by WHO.

Study conducted at Agakhan University in Karachi, Pakistan, 1992 showed that with the threshold of 140mg %, 8.6% of screened population had abnormal glucose values⁽³³⁾.

7) GLYCATED BLOOD PROTEINS

a) Glycosylated Hemoglobin (HbA1c)

Glycated hemoglobin is the linkage between glucose and hemoglobin. Glycosylated Hb is a measure of the control of glycemia over the previous 2 to 3 months. It may be useful only in the retrospective documentation of GDM.

In pregnant women with GDM, there is fasting hypoglycemia, increased erythropoiesis, less glycated Hb and fluctuations in blood glucose level due to rapid change in hormonal milieu. So HbA1c is not much effective. In normal women it is 5.7% whereas HbA1c>8.8% is suggestive of GDM. Level is reduced significantly in patients with anemia. It yields false positive results in 41% and false negative in 26%⁽²⁰⁾.

Limitations

- 1) It cannot be used for day to day control (Irwin)
- 2) It cannot register hypoglycemia (Irwin)

Advantages

1. In pregestational diabetes, to know the retrospective blood glucose control at the time of conception.
2. Monitoring the control of glucose in established diabetic pregnancy.

b) Glycosylated Albumin

Glycosylated albumin has a shorter half life than HbA1c and may be more effective in identifying women with an acute rise in mean day blood glucose.

It reflects the blood glucose status over the preceding 1-3 weeks (Kennedy et al 1984).

8) FRUCTOSAMINE

Glucose condenses non enzymatically with the terminal aminogroup of serum proteins to form an unstable aldamine. This undergoes an Amadori rearrangement to form more stable ketoamine. This is termed fructosamine because of structural similarity to fructose. It assesses the short term state of glycemic control over the past 2 weeks⁽⁷⁾. In study by *Roberts et al*⁽²¹⁾ it was concluded that Fructosamine has a sensitive of 50%. Hence it is not an useful screening test.

DIAGNOSTIC TEST

Various diagnostic test for GDM are :

- 1) O-Sullivan And Mahan's OG TT Test
- 2) WHO 75 gm OG TT
- 3) Intravenous Glucose Tolerance Test

1) O-SULLIVAN AND MAHAN OG TT TEST

This test served as a gold standard for the diagnosis of gestational diabetes mellitus. In O Sullivan Studies whole blood was tested using the O Somogyi Nelson method which measured reducing substances also. Plasma glucose values is 14% higher than those in whole blood obtained from the same sample using the same assay method. NDDG reinterpreted O Sullivan's data by glucose oxidase method on plasma by increasing O'Sullivan's criteria by 15%. This criteria reaffirmed by ADA in 1985 stated that the glucose oxidase method is specific for glucose and generally result in 5mg /dl decrease in measured values (Carpenter and Coustan).(23)

The test should be made in the morning after an overnight fast of 8 hours after 3 days of normal diet and physical activity. The subject should remain seated and not stand or smoke throughout the test.

DIAGNOSTIC CRITERIAS FOR GDM

	Glucose Load		Fasting (mg%)	1 hr (mg%)	2 hr (mg%)	3 hr (mg%)
O Sullivan and Mahan(56)	100gm	Whole blood	90	165	145	125
Carpenter & Coustan(58)	100gm	Plasma	95	180	155	140
NDDG(57)	100gm	Plasma	105	190	165	145
Langer et al	100gm	Plasma	105	190	165	145

Sacks	75gm	Plasma	95	180	155	-
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When two values are met or exceeded then a positive diagnosis is made.

2)WHO 75 gm OG TT

In many parts of the world⁽⁶³⁾ 2 hour 75 gm OGTT is used during pregnancy. Criteria adopted by WHO for diagnosing GDM is shown in the table below.

	Fasting	2 hrs PPG
IGT	< 126	140-200
DM	> 126	> 200

If the abnormal 2 hour value is 140-200 mg% it is called impaired glucose tolerance. The impaired glucose tolerance by WHO in pregnancy is GDM.

Metzger et al reported that fasting blood glucose after an over night fast was approximately 10mg% low in pregnant than in non pregnant level. So fasting plasma glucose is not diagnostic of GDM. This one step procedure of diagnosing GDM using WHO OGTT serves dual purpose of both screening and diagnosing GDM⁽²²⁾. WHO OGTT in pregnancy uses the same threshold as in non pregnant individuals.

Advantages:

- 1) Load on the lab is reduced, since number of blood samples drawn are reduced
- 2) Patient's compliance is increased

Disadvantages:

It requires patient to be in the fasting state.

3) INTRAVENOUS GLUCOSE TOLERANCE TEST

This test was adopted for use in pregnancy by Silverstone et al ^(24,25). Intravenous glucose is given at the dose of 0.5g/kg as 25% or 50% solution and blood glucose is estimated every 10 minutes for a period of 60 minutes. The unit of measurement of the test, the K value is a measure of the percentage decrease of glucose over time, $K = 70 (1/2) t$.

Criteria for selection by Silverstone ⁽²⁴⁾

Subjects with normal fasting blood sugar, free of glucosuria without any family history of diabetes, history of macrosomia, liver disease, cardiac disease or hypertension. They were not on any drugs that affect the glucose tolerance.

If K value is < 1.5 in pregnant women, it means that she is diabetic.

None has performed the test in a large group of population and none has related the test results to perinatal morbidity.

Advantages

- 1) Unaffected by variation in gastric emptying
- 2) It is useful when one vomits with oral test.

Disadvantages

- 1) More expensive
- 2) Not subjected to the normal influence of gastrointestinal physiology.

Screening Strategy for Gestational Diabetes Mellitus

In Fourth⁽³⁾ International Workshop conference for diabetes recommendations were made for selective screening based on risk assessment.

Blood glucose testing is not required in low risk. In women with average risk blood glucose testing is done at 24-28 weeks. In high risk patients blood glucose is tested as soon as feasible and if it is negative the test is repeated again at 24-28 weeks and again in the III trimester.

High Risk :

- 1) Member of ethnic group with high prevalence of diabetes.
- 2) Age more than 25 years
- 3) Known diabetes in first degree relative
- 4) Weight abnormal before pregnancy
- 5) History of abnormal glucose metabolism.
- 6) History of poor obstetric outcome.

Low risk

If all the above risk factors are absent.

Average Risk

Women of Hispanic, African, Native American, South or East Asian Origin.

Blood glucose testing is done as 2 step procedure. First 50g oral glucose challenge test is followed by diagnostic 100gm oral glucose tolerance test, if

the results exceed a predetermined plasma glucose concentration. It can be done as a single step procedure also.

For a large population where the prevalence of GDM is high, universal screening for GDM is required. When both oral glucose challenge test and diagnostic OGTT for GDM has to be done for the huge population

- i) It will be cumbersome
- ii) The phenomenon of no show will be there
- iii) Load on the lab will be more
- iv) Cost of the test will be high

Ideal diagnostic test must be cheap, reproducible and have high specificity. Pregnant women often experience nausea while in the fasting state. They have to travel to the clinic and wait an additional 2 hr before eating. WHO OGCT uses the same glucose load and has the same cut off value as WHO OGTT. WHO OGCT has the advantage of patient coming in any feeding state and it can be done on the same day when the patients report to the antenatal clinic. Hence why WHO OGCT with the above said advantages can't be used as a diagnostic test for GDM ?

AIM OF THE STUDY

- To find out whether 2 hours 75g WHO OG CT is as efficacious as 2 hours 75g WHO OGTT in detecting gestational diabetes mellitus in antenatal population between 16 – 32 weeks.
- To analyse fetal outcome in GDM patients.

MATERIALS AND METHODS

Place of Study : Institute of Obstetrics and Gynaecology,
Egmore.

Year of Study : 2005 – 2006.

Nature of Study : Prospective Study

Selection of Cases : 800 pregnant women between 16 – 32 weeks
were randomly Selected irrespective of
parity, age Previous obstetric outcome.

Inclusion Criteria :

1. All the women with gestational age by history and clinical examination between 16 to 32 weeks were included in this study.
2. Women who were not sure of the last menstrual period and whose clinical examination was inappropriate had an ultrasound examination and when the period of gestation was between 16 – 32 weeks were included in this study.

Exclusion Criteria :

1. Women who were not within 16-32 weeks of gestation were excluded from the study.
2. Women who were already pregestational diabetes were excluded.

Method

Pregnant women who were selected with the above criteria had urine glucose and albumin estimation done. Complete and careful clinical history was taken with regard to menstrual cycles, previous obstetric history, family history. Complete clinical examination was done. Ultrasound examination was done to rule out congenital anomalies, to detect hydramnios and macrosomia. Patients were subjected to WHO OGCT. 75g glucose was given in 200ml of water. 2 hour later 1 cc of venous blood was drawn in a test tube containing EDTA and immediately sample was taken for plasma glucose analysis. Following the glucose drink patients were prohibited from eating food or drinking except water.

Same patients were instructed to come 2 days later after regular diet consumption with over night fasting of 8 hours and 2 hrs 75g WHO OGTT was done. The sensitivity and specificity of WHO OGCT in diagnosing GDM is estimated by statistical analysis of plasma glucose values.

Plasma glucose was estimated by *enzyme method* in our hospital. The method used in our hospital is described below :

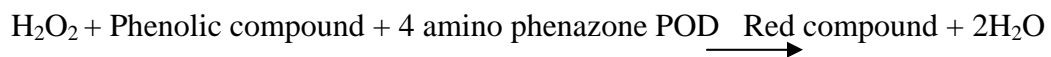
ENZYME METHOD

To 20µl of plasma 2ml of the reagent containing glucose oxidase is added and kept in an incubator at 37°C for 15-20 minutes. The final colour developed in this test sample is compared with the colour developed in the standard sample assayed by the same method in calorimeter at 520nms. The plasma glucose value

is determined by the intensity of the red colour developed by comparing with the standard sample.

Chemical Reaction

Glucose is oxidized by glucose oxidase into gluconic acid and H₂O₂. H₂O₂ in the presence of peroxidase oxidises the 4 amino phenazene and phenolic compound to a red coloured compound.



(H₂O₂ : Hydrogen Peroxide, H₂O : Water)

RESULTS AND ANALYSIS

Total number of patients screened for gestational diabetes mellitus were 800. Out of this, 87 were diagnosed as GDM using WHO criteria.

TABLE – 1
AGE DISTRIBUTION

Age (yrs)	Non-GDM Patients	%	GDM Patients	%
17-20	96	13.46	Nil	Nil
21-25	140	19.63	11	12.64
26-30	373	52.31	36	41.37
31-35	81	11.36	31	35.63
36-40	23	3.26	9	10.34

41.37% of GDM patients were in the age group of 26-30 years.

TABLE – 2
AGE

Age	Total Patients	GDM Patients	%
17-20	96	Nil	Nil
21-25	151	11	7.28
26-30	409	36	8.8
31-35	112	31	27.68
36-40	32	9	28.13

As age advances the prevalence of GDM increases in our study.

Mean \pm SD in GDM group = 32.50575 \pm 2.555767.

Mean \pm SD in non GDM group = 26.59327 \pm 4.125157

P = 0.0 significant

There exist a statistically significant difference in age between GDM and Non GDM group.

TABLE – 3 (a)
GRAVIDA

Gravida	Non-GDM patients	%	GDM Patients	%
G1	401	56.24	31	35.63
G2	191	26.79	26	29.88
G3	90	12.62	19	21.83
G4	26	3.65	10	11.49
G5	3	0.42	1	1.15
G6	2	0.28	Nil	Nil

35.63% of GDM patients were found to be primis.

Table – 3(b)

Gravida	Total Patients	GDM Patients	%
G1	432	31	7.18
G2	217	26	11.98
G3	109	19	17.43
G4	36	10	27.78
G5	4	1	25
G6	2	Nil	Nil

As the number of gravida increases the prevalence of GDM increases. (p = 0.0) significant.

TABLE – 4
EDUCATIONAL STATUS

Educational Status	Non-GDM Patients	%	GDM Patients	%
Illiterate	273	38.29	20	22.99
Primary Education	186	26.08	21	24.14
High School Education	195	27.34	33	37.93
College Education	59	8.27	13	14.94

37.93% of GDM patients had high school education in our study.

TABLE – 5
SOCIOECONOMIC STATUS

Socioeconomic Status	Non-GDM Patients	%	GDM Patients	%
I	Nil	Nil	Nil	Nil
II	9	1.26	6	6.9
III	104	14.59	19	21.84
IV	272	38.15	27	31.03
V	318	44.6	35	40.23

In our study 35 GDM patients belonged to grade V socioeconomic status and 27 GDM patients belonged to Grade IV socioeconomic status..

TABLE – 6 (a)
BMI

BMI	Non-GDM Patients	%	GDM Patients	%
≤ 20	48	6.73	5	5.74
21-24	446	22.55	21	24.13
25- 29	140	19.63	34	39.08
≥ 30	79	11.08	27	31.03

In our study BMI ≤ 20 was seen in 5.74% of GDM patients and BMI ≥ 30 (obese) was seen in 31.03% of GDM patients.

TABLE – 6 (b)

BMI	Total Patients	GDM Patients	%
≤ 20	53	5	9.43
21-24	467	21	4.5
25- 29	174	34	19.5
≥ 30	106	27	25.47

The incidence of GDM in BMI ≥ 30 was 25.47%. (p = 0.0 significant)

TABLE – 7 (a)
FAMILY HISTORY

Family History	Non GDM Patients	%	GDM Patients	%
Positive	101	14.17	44	50.57
Negative	612	85.83	43	49.43

50.57% of GDM patients had positive family history of diabetes mellitus in our study.

TABLE – 7 (b)

Family History	Total Patient	GDM Patients	%
Positive	101	44	43.56
Negative	612	43	7.02

Incidence of GDM in patients with positive family history of diabetes was 43.56%. (p = 0.0 significant).

TABLE – 8
GESTATIONAL AGE AT TESTING

G.A. at Testing	Non GDM Patients	%	GDM Patients	%
16-20 wks	100	14.02	7	8.05
21-24 wks	154	21.6	17	19.54
25-28 wks	196	27.49	49	56.32
29-32 wks	265	35.48	14	16.09

Out of 87 patients 7 patients were diagnosed between 16-20 weeks, 17 patients were diagnosed between 21-24 weeks, 49 patients were diagnosed between 25-28 weeks, 14 patients were diagnosed between 29-32 weeks. Out of 100 patients in non GDM group in 16-20 weeks 51 reported for assessment of plasma glucose at 24-28 weeks and 30 reported for assessment of plasma glucose at 32 weeks and were found to be negative. Out of 154 patients in non GDM group in 21-24 weeks 100 reported for assessment at 28 weeks (Out of 100, 2 were found to be positive)and 57 reported for assessment at 32 weeks. Out of 196 patients who were negative at 24-28 weeks, 97 came for assessment at 32 weeks and were negative. (p = 0.0 significant)

TABLE – 9
RISK FACTORS IN PREVIOUS PREGNANCY

Risk factors	Non GDM patients	%	GDM Patients	%
H/o Spontaneous abortion	72	14.72	20	22.99
H/o. Macrosomic baby	15	2.1	8	9.2
H/o. Sudden IUD	16	2.24%	5	5.75
H/o. unexplained still birth	18	2.52	7	8.04
H/o. unexplained neonatal death	9	1.26	5	5.75
H/o GDM	6	0.841	2	2.3
H/o PIH	35	4.91	6	6.89
H/o Congenital Anomalies	3	0.42	4	4.6

Among GDM patients 20 had history of spontaneous abortion, 8 had history of delivery of macrosomic baby, 5 had sudden IUD, 7 had history of unexplained neonatal death, 2 had history of GDM, 6 had history of PIH and 4 had history of babies with congenital anomalies (4VSD, 1 anencephaly, 1 meningocele) in previous pregnancies.

TABLE – 10
RISK FACTORS IN PRESENT PREGNANCY

Risk factors	Non GDM Patients	%	GDM Patients	%
Obesity	79	11.08	27	31.03
Recurrent UTI	20	2.81	11	12.64
Moniliasis	17	2.38	23	26.44
PIH	51	7.15	14	16.09
Hydramnios	11	1.54	2	2.3
Glycosuria	83	11.64	9	10.34
Cong. Malformation	8	1.12	1	1.15
Macrosomia	36	5.89	18	20.69

Among GDM patients 27 were obese, 11 had history of recurrent UTI, 23 had history of moniliasis, 14 had history of PIH, 2 had hydramnios, 9 had glycosuria, 1 had congenital anomaly (VSD) and 18 had macrosomia.

TABLE – 11
PLASMA GLUCOSE

Plasma Glucose	WHO OGTT	%	WHO OGCT	%
61-70	63	7.88	59	7.38
71-80	71	8.88	70	8.75
81-90	103	12.88	108	13.5
91-100	116	14.5	120	15.75
101-110	140	17.5	130	16.25
111-120	95	11.88	98	12.25
121-130	97	12.13	99	12.38
131-139	28	3.5	29	3.63
140-150	21	2.63	24	3
151-160	22	2.75	18	2.3
161-170	19	2.38	22	2.75
171-180	14	1.75	13	1.63
181-190	11	1.38	10	1.25

The prevalence of GDM in our study was 10.89%. All the patients who were diagnosed as GDM by WHO OGTT had positive values by WHO OGCT also

	Mean \pm SD of plasma glucose values		
	GDM	Non-GDM	P value
WHO OGTT	162.0805 \pm 13.01271	99.26648 \pm 13.01271	0.0 significant
WHO OGCT	162.03482 \pm 13.03482	99.90182 \pm 20.20579	0.0 significant

Positive patients were referred to pregnancy diabetic service of IOG for treatment of GDM and followed up until delivery.

Out of 87 GDM patients only 65 patients came for delivery at IOG. Fetal outcome were assessed for these patients.

TABLE – 12
MODE OF DELIVERY

Mode of delivery	GDM Patients	%
Normal	24	36.92%
Assisted Vaginal	9	13.84%
Caesarean section	32	49.23%

Babies were delivered by caesarean section in 49.23% of GDM patients.

FETAL OUTCOME OF GDM PATIENTS

TABLE – 13 (a)
GESTATIONAL AGE AT DELIVERY

Gestational Age at Delivery	GDM Patients	%
Preterm	9	13.85%
Term	56	86.15%

Preterm delivery was seen in 13.85% of GDM patients and term delivery was seen in 86.15% of GDM patients.

TABLE – 13 (b)
BIRTH WEIGHT

Birth Weight (kg)	GDM patients	%
2 – 2.4	5	7.69
2.5 – 2.9	12	18.46
3 – 3.4	18	27.69
3.5 – 3.9	14	21.54
≥ 4	16	24.61

Macrosomia defined as birth weight ≥ 4 kg was seen in 24.61% of GDM pregnancies delivered at IOG.

TABLE – 13 (c)
APGAR SCORE

Apgar Score	GDM Patients	%
8 and above	51	78.46%
5 – 7	11	16.92%
1 – 4	3	4.61%

Apgar score of 8 and above was seen in 78.46% of GDM patients in our study. Apgar score of 5-7 was seen in 16.92% of GDM patients,

TABLE – 13 (d)
NEONATAL COMPLICATIONS

Neonatal Complications	GDM Patients	%
Hypoglycemia	19	29.23%
Hypocalcemia	13	20%
Hyperbilirubinemia	10	15.38%
Congenital Anomalies	1	1.54%
RDS	6	9.2%

Hypoglycemia was seen in 29.23%, hypocalcemia was seen in 20% and RDS in 9.2% of infants of GDM.

PERINATAL MORTALITY

All the women with GDM who delivered at IOG had good fetal outcome. Even the babies who had complications recovered well.

DISCUSSION

A prospective study was conducted in IOG during 2005- 2006. For this study 800 randomly selected pregnant women in 16- 32 weeks of gestation were included. 2 hour 75g WHO OGCT and 2 hr 75g WHO OGTT were estimated in all these women. This study was designed to find out whether 2 hr 75g WHO OGCT is as effective as 2 hr 75g WHO OGTT in diagnosing GDM.

PREVALENCE

According to *Beischer et al* 1991, the prevalence of GDM in Indian subcontinent is 15%.

In a study conducted at Jean Verdier Hospital, France it was identified that prevalence of GDM was 15.65% , if universal screening method is adopted⁽²⁸⁾.

In a study conducted by *Dr. Anjalakshi* for evaluation of diagnostic criteria for AGT in South Indian Pregnant women prevalence of GDM was found to be 15% ⁽²⁶⁾.

Seshiah et al found the prevalence of GDM to be 16.2% in India in his study using WHO Criteria⁽²²⁾. *Schmidt et al* in 2001 found out the incidence of GDM to be 7.2% by WHO criteria⁽²⁷⁾.

In our study prevalence of GDM by WHO criteria was 10.89%.

PREVALENCE OF GDM

Study	Prevalence
Beischer et al	15%
Benchimol et al	15.65%
Dr. Anjalakshi	15%
Seshiah et al	16.2%
Schmidt et al	7.2%
Our Study	10.89%

WHO OGTT

Seshiah et al found that out of 891 pregnant women who underwent 50g GCT and a subsequent 75g OGTT 144 had GDM by WHO criteria. Out of these 144 cases only 113 had initial 50g value > 130 mg/dl. So diagnosis of GDM by OGTT based on initial GCT screening leaves 21.3% undiagnosed. The two step procedure is not practical as patients have a phenomenon of no show and number of blood samples are more thus increasing the load on the lab. So it is ideal to go for WHO OGTT which detects more cases of GDM, reduces load on the lab, improves the patients compliance⁽²²⁾. In the present study those who had normal OGTT in the earlier pregnancy were asked to review later for reassessment of plasma glucose by WHO criteria and there was a phenomenon of no show in these

patients when they were asked to come later for reassessment of plasma glucose similar to *Prof. V. Seshiah's* study.

Schmidt et al compared ADA criteria and WHO criteria and found that among 4877 women studied, 2.4% presented with GDM by ADA criteria and 7.2% by WHO criteria. GDM by WHO predicts an increased risk of macrosomia, preeclampsia and perinatal death⁽²⁷⁾.

Benchimol et al in his study at Jean Verdier hospital used one step WHO OGTT and found the prevalence of GDM to be 15.65%⁽²⁸⁾.

To standardize the diagnosis of GDM WHO has proposed using a 2 hour 75 g OGTT with a threshold plasma glucose concentration greater than 7.8mmol/l at 120 minutes. These criteria identify two to three times more women as having GDM than American methods⁽²⁹⁾. Prevalence of GDM in our present study is 10.89%.

In **HAPO study** which is a 5 years prospective observational study recruiting 25000 pregnant women in 10 countries uses 2 hrs 75g OGTT using WHO criteria. One can understand the importance of WHO OGTT in diagnosing GDM and predicting adverse pregnancy outcome when such a large study adopts the WHO criteria⁽³⁰⁾. In our study also we have used WHO criteria for diagnosing GDM.

Since all the above studies use WHO OGTT for diagnosing GDM and have detected a large number of cases, WHO OGTT was used as a gold standard for comparing WHO OGCT.

WHO OGTT Vs WHO OGCT

A study was done by David J Petitt by comparing the one step WHO OGCT and NDDG criteria. He showed that the subjects who were positive by NDDG criteria were also positive by WHO OGCT and it identifies more cases which were considered normal by NDDG criteria³¹.

There is strong argument for not requiring an overnight fast before the initial glucose tolerance test. Pregnant women often experience nausea when fasting and may refuse or be unable to fast until the test. It may be impossible for the women to get up, travel to the clinic and then wait an additional 2 hrs before eating. Because glucose concentration during the oral glucose tolerance test is affected little by the time since the last meal⁽³²⁾, fasting adds very little to the glucose tolerance test making the test more cumbersome. The nonfasting 2 hrs post 75g glucose concentration strongly predicts adverse outcome for the mother and off spring. O' Sullivan and Mahan's OGTT predicts the development of diabetes in the mother later on and it doesn't give the fetal outcome. But WHO criteria which is used in our study predicts both maternal and fetal outcomes.

In our study WHO OGCT was done in 800 cases on whom WHO OGTT was also done. It was found that all patients who were detected to have GDM by WHO OGTT were also positive by WHO OGCT. Hence in our study it is shown that WHO OGCT is as efficacious as WHO OGTT in diagnosing GDM. WHO OGCT also predicts adverse maternal and fetal outcomes.

A study was done by Dr. Anjalakshi on abnormalities of glucose intolerance in pregnant women by comparing WHO OGCT with WHO OGTT. Sensitivity and specificity was found to be 0% and 84% respectively. This was in contrast to our study which showed sensitivity and specificity of 100%.

	Sensitivity	Specificity
Dr. Anjalakshi	0%	84%
Our Study	100%	100%

In our study WHO OGCT is as efficacious as WHO OGTT in detecting GDM. More studies are needed before its clinical application can be made to diagnose GDM.

STATISTICAL DATA		
WHO OGCT	+	WHO OGTT
		+
		-
		87
		0
		a
		b
		0
		713
		c
		d

$$\text{Sensitivity} = \frac{87}{87} \times 100 = 100\%$$

$$\text{Specificity} = \frac{713}{713} \times 100 = 100\%$$

MATERNAL OUTCOME

AGE :

Age	Total Patients	GDM Patients	%
17-20	96	Nil	Nil
21-25	151	11	7.28
26.-30	409	36	8.8
31-35	112	31	27.68
36-40	32	9	28.13

Coustan et al⁽³⁶⁾ found that the incidence of GDM is 3.8% in women of 30 – 34 years of age but only 0.7 in women younger than 21 in a large study of unselected population.

In our study the incidence of GDM is 27.68% in women of 31-35 years. It is found that in our study as age advances prevalence of GDM increases.

Moses et al⁽³⁷⁾ found that age more than 30 years was present in 8.5% of GDM patients and younger than 21 years was present in 0.7% of GDM patients.

Seshiah et al⁽²²⁾ in his study in 2005 found that the incidence of GDM in < 20 years is 14.5%, 20-24 years 13.7%, 25-29 years 19.5%, \geq 30 years 25%. It is

shown that as the age advances prevalence of GDM increases which is comparable to our study. In our study ($p = 0.0$) there exists a statistically significant difference in age between the GDM and the non-GDM group.

GRAVIDA

Prevalence of Gestational Diabetes increases with gravidity from 16.3% in primi to 25.8% in gravidas ≥ 4 in a study by *Seshiah et al*⁽²²⁾.

In our study the prevalence of gestational diabetes increased with gravidity from 7.18% in primi to 27.78% in gravida 4. ($p = 0.0$ and it is statistically significant) and it is similar to the finding in Seshiah's study.

Pyke De et al found that the incidence of gestational diabetes increases with parity⁽³⁸⁾.

BMI

Serirat et al⁽³⁹⁾ in 1992, in a study found that obesity was present in 26.5% of patients with GDM. In our study BMI ≥ 30 was presenting 31.03% of patients with GDM. ($p=0.0$ and it is statistically significant)

In a study by *Seshiah et al*⁽²²⁾ the incidence of GDM was 33.3% in patients with BMI ≥ 30 . The incidence of GDM increases as the BMI increases, comparable to our study..

FAMILY HISTORY

Serirat et al in 1992 has shown that family history of diabetes is present in 23.1% of patients with abnormal glucose tolerance⁽³⁹⁾.

Moses et al has shown that family history of diabetes is present in 11.6% of patients with GDM⁽³⁷⁾.

In our study family history of diabetes was present in 50.57% of patients with GDM. (p = 0.0)

PIH

Suhoven & Terano et al⁽⁴¹⁾ in 1993 reported the incidence of PIH and preeclampsia to be 2 times more common among GDM patients than controls. (19.8% Vs 10%).

In a study by *Schmidt et al*⁽²⁷⁾, it was found that frequency of preeclampsia was 5%

PIH was seen in 13.7% in GDM in a study by *Cousins et al*⁽⁴⁰⁾.

In our study PIH was present in 16.09% of patients with GDM which was equivalent to the finding by Suhoven.

Study	Incidence of PIH in GDM
Suhoven et al	19.8%
Schmidt et al	5%
Cousins et al	13.7%
Our study	16.09%

HYDRAMNIOS

Rosenn and Combs et al found the incidence of hydramnios to be 26.4% in their study⁽⁴³⁾.

Biggio et al in 1999 reported that hydramnios occur in 20% of diabetic pregnancies.

In Cousins study Incidence of hydramnios ranges from 2 to 2.1% in GDM similar to our study⁽⁴⁵⁾.

Study	Incidence of Hydramnios in GDM
Rosenn et al	26.4%
Cousins et al	2-2.1%
Our study	2.3%

GESTATIONAL AGE AT TESTING

A study was done by *Seshiah et al* for detection of GDM in the three trimester of pregnancy. Among the studied patients 16.3% were within 16 weeks of gestation, 23.1% were between 17-23 weeks, 60.6% were more than 24 weeks(64).

In our study involving screening and diagnosing GDM in antenatal population between 16-32 weeks, 27.59% of GDM patients were between 16-24 weeks and 72.41% of GDM patients were between 25-32 weeks.

MATERNAL MORTALITY

Maternal deaths have become rare in women with diabetes although as emphasized by Cousins who stated that mortality is increased 10 folds most often as a result of ketoacidosis, hypertension, preeclampsia, pyelonephritis and patients with coronary artery disease.

In our study there was no maternal mortality.

FETAL OUTCOME

Birth Weight

Spellacy, WN Miller, S. Winegar found that Macrosomia was present in 50% of pregnant patients with GDM⁽⁵⁰⁾.

In our study macrosomia was seen in 20.69% of pregnant patient with GDM.

Langer et al also found 50% of pregnant patient with GDM to have macrosomia⁽⁵¹⁾.

NEONATAL COMPLICATIONS

Hypoglycemia

Cowett et al found that infants of women with GDM have an incidence of neonatal hypoglycemia that approaches 30-50%⁽⁵³⁾.

According to James the frequency of hypoglycemia is 18-49%⁽⁵²⁾. Hypoglycemia was seen in 29.23% of infants of GDM in the present study. This was same as the above studies.

Study	Incidence of hypoglycemia in infants of GDM
Cowett et al	30-50%
Our Study	29.23%

HYPOCALCEMIA

Hypocalcemia defined as total serum calcium level less than 7mg/dl affects 50% of infants with GDM (*Marshall et al*)⁽⁵⁴⁾

In our study hypocalcemia was seen in 20% of infants with GDM.

HYPERBILIRUBINEMIA

Tyralla et al states that hyperbilirubinemia defined by serum bilirubin level > 13mg/dl was seen in 20% of GDM pregnancies⁽⁶⁰⁾.

Hyperbilirubinemia was present in 15.38% of GDM pregnancies in our study which was comparable similar to *Tyralla et al* study.

Study	Hyperbilirubinemia % in infants of GDM
Tyralla et al	20%
Our Study	15.38%

RESPIRATORY DISTRESS SYNDROME

Robert et al found that the risk of developing RDS was 5.67% in infants of GDM mothers⁽⁶¹⁾.

In our study RDS was present in 9.2% of GDM pregnancies, probably due to the high caesarean section in GDM.

SUMMARY

- 1) In 800 Randomly selected patients who attended our antenatal clinic prevalence of GDM was found to be 10.89% by WHO criteria.
- 2) 2 hr 75g WHO OGCT was performed on all 800 patients attending our antenatal clinic between 16-32 weeks since ethnically Indian belong to a high risk group.
- 3) Out of 100 non GDM patients between 16-20 weeks only 51 reported reassessment at 24-28 weeks and 30 reported for reassessment at 32 weeks. Out of 154 non GDM patients at 21-24 weeks, 100 reported for reassessment at 24 weeks and 57 reported for reassessment at 32 weeks. Out of 196 non GDM patients at 25-28 weeks only 97 came for reassessment at 32 weeks. This demonstrates the phenomenon of no show.
- 4) In our study it was found that all the patients who were diagnosed as GDM by WHO OGTT gave positive results with WHO OGCT. Thus sensitivity and specificity of WHO OGCT is 100%. WHO OGCT is useful as a diagnostic test for GDM. No show is not possible in first visit.

- 5) In our study it was found that i) As age increases the prevalence of GDM increases, ii) As number of gravida increases prevalence of GDM increases, iii) As BMI increases prevalence of GDM increases, iv) Positive family history predicts the higher risk of developing GDM, v) Macrosomia was seen in 20.69% of GDM patients.

Complications	% in GDM Patients
Hydramnios	2.3%
PIH	16.09%
Macrosomia	20.69%
Caesarean section rate	49.23%
Hypoglycemia	29.23%
Hypocalcemia	20%
Hyperbilirubinemia	15.38%
RDS	9.2%

Among mothers whose babies had macrosomia 13 were on insulin and 5 were on medical nutrition therapy.

- 6) Hence early detection of gestational diabetes and effective management to maintain optimal blood glucose levels will drastically reduce maternal morbidity due to gestational diabetes and bring about a definite reduction in perinatal mortality rate.

CONCLUSION

- 1) Screen pregnant women universally for gestational diabetes.
- 2) 2 hr 75g WHO OGCT can be used to diagnose GDM and is as efficacious as 2 hr 75g WHO OGTT. More studies are needed before clinical application can be made.
- 3) To detect Gestational Diabetes Mellitus early.
- 4) In spite of treatment 20.69% of GDM patients had macrosomic babies. In infants of GDM patients RDS was seen in 9.2%, hypoglycemia was seen in 29.23%, hyperbilirubinemia was seen in 15.38%. So more stringent treatment of GDM will improve perinatal outcome.

Screening universally for GDM improves the perinatal outcome, postpones the onset of diabetes in mother and also has a role in primary prevention of diabetes in offspring.

BIBLIOGRAPHY

- 1) Davidsons Principle and Practice of Medicine, 19th edn.
- 2) Carbohydrate metabolism and Gestational Diabetes == Catalano PM clinic Obstetrics and Gynaecology, 1994, March 37(1) 25-38.
- 3) Williams Obstetrics, 22nd edn.
- 4) Gestational Diabetes - Criteria for Screening and Diagnosis - Santini – C Dravdi – Maraldi – C – Minerva. Endocrinology 1994. June ; 19 (2); 57-61.
- 5) Fernando Arias. Practical guide to high risk pregnancy and delivery, 2nd end.
- 6) Harrison's Principle of Internal Medicine. 16th edn.
- 7) Professor V. Seshiah; Diabetes and Pregnancy.
- 8) Frienkel N. Effects of conceptus on maternal metabolism during pregnancy – P. 679, ZN Lethal BS Wrenshall GA (Eds) On the nature and treatment of Diabetes – Exerta Medical Amsterdam 1965.
- 9) Fuhrmann K. Reiter H, Semmlar K et al. Prevention of congenital malformation in infants of insulin dependent diabetic mothers. Diabetes Care 6-219, 1983.
- 10) Beischer NA. Oats JN, Henry OA et al. Incidence and severity of Gestational Diabetes Mellitus according to country of birth in women living in Australia. Diabetes Suppl. 2, 1991.

- 11) Green JR, Pawson JG, Schumacher IB. et al. Glucose tolerance in Pregnancy. Ethnic variation and Influence of body habitus. Am. J. Obstet. Gynaecol. 163; 86, 1990.
- 12) Metzger BE : Organising committee summary and recommendation of the Third International Workshop conference on gestational diabetes mellitus. Diabetes suppl. 2, 40; 197, 1991.
- 13) Coustan DR. Diagnosis of gestational Diabetes: Are new criteria needed ? Diabetes Rev. 1995, 3 : 614 – 620.
- 14) The expert committee on the diagnosis and classification of Diabetes Mellitus: Report of the expert committee on the diagnosis and classification of Diabetes Mellitus. Diabetes care 1997; 20 – 183-1197.
- 15) Berkowitz G.S. Lopinshi RH, Wein R. Lee D : Race / Ethnicity and other risk factors for gestational diabetes. Am J. of epidemiol. 135 (9). 965 – 73, 1992, May I.
- 16) Michael De Swiet. Medical disorders in Obstetric Practice. 4th edn.
- 17) Sacks DA, Greenspoon, Fotheringham N. Could the fasting plasma glucose assay be used to screen for gestational diabetes ? J. Reprod. Med. 1992; 37; 907 – 9.
- 18) Daniele P, Fischer U, Spinass GA et al. Using fasting plasma glucose concentrations to screen for GDM. A prospective population based study. BMJ 1993, 319 : 812 – 5.

- 19) Metzer BE, Kim YL. Detection and diagnostic strategies for gestational diabetes mellitus. M. Jovanovic L Carlo Di Renzo G et al (eds) Text book of diabetes and pregnancy. London. Taylor and Francis. 2003 : 173.
- 20) Cousin SL et al Glycosylated Hb as a screening test for carbohydrate intolerance in pregnancy. Am J Obst Gynaecol 1984; 150: 455-60.
- 21) Robert AB, Baker JR. Fructosamine in Diabetic Pregnancy. Lancet 1983; 998 – 1000.
- 22) V. Seshiah, V. Balaji, Madhuri S Balaji, One step procedure for screening and diagnosing for gestational diabetes mellitus. The journal of obstetrics and Gynaecology of India. Vol. 55, 6, Nov/Dec. 2005, pg. 523-529.
- 23) Diabetes in pregnancy. Clinical Obstetrics and Gynecology. 2000.
- 24) Silverstone FH. Solomons E. Rubicius J. The rapid intravenous glucose tolerance test in pregnancy. Indian J. Clin. Invest. 40 : 2180; 1961.
- 25) Posner NA, Silverstone FH. Brewer J. Heller M. Simplifying the intravenous glucose tolerance test J. Reproductive Medicine 27 ; 633, 1982.
- 26) Dr. Anjalakshi Chandrasekhar PhD thesis. Evaluation of diagnostic criteria for abnormal glucose tolerance in South Indian pregnant women (2002).
- 27) Maria. I. Schmidt, Bruce. B Duncan, Angela J. Reichelt. Gestational Diabetes Mellitus Diagnosed with a 2 hr 75g oral glucose tolerance test and adverse pregnancy outcomes. Diabetes care 2001.

- 28) Benchimol M, Cassone, Faure C, Carbillon L, comparison of two routine screening strategies for gestational diabetes mellitus. The experience of Jean Verdier Hospital. *Gynaecol Obstet Fertil* 2006 Feb; 34(2) : 107 – 114, E pub 2006, jan.
- 29) Text book of diabetes 1 and 2 – John C. Pickup and Gareth Williams (3rd Edition).
- 30) *International journal of Gynecol Obst.* 2002, july; 78(1);69-77.
- 31) David J Pettitt, PH Bennett, RL Hanson. Comparison of WHO and NDDG procedure to detect abnormalities of glucose tolerance during pregnancy. *Diabetes care* 1994.
- 32) Gough WW, Shack MJ, Bennett PH, Burch TA, Miller M. Evaluation of Glucose in Pima Indian by longitudinal studies (Abstract) *Diabetes* 19, suppl. (1), ; 388, 1970.
- 33) Rizvi JH Rasul, S Malik, S. Rehamatullah, A. Khan , M.A. Dept. of Obst. And Gynaecol, Agakhan University, Karachi, Pakistan. Experience in screening for abnormal glucose tolerance test in pregnancy. Maternal and perinatal outcome. *Asia Oceania Journal of Obstet and Gynaecol* ; 18(2) ; 99-105 (1992 june).
- 34) Nahum GG, Wilson SB, Stanislaw H : Early pregnancy glucose screening for GDM. *J Reprod Med* 2002; 47 (8) : 656 – 662.
- 35) E.cosson et al : Screening and insulin sensitivity in gestational diabetes. Abstract volume of the 40th Annual meeting of the EASD, September 2004. A 350.

- 36) Coustan DR, Nelson C, Carpenter MW et al. Maternal Age and screening for Gestational Diabetes, a population based study. *Obst. Gynaecol* : 73:557; 1989.
- 37) Moses R et al. Gestational Diabetes – Do all women need to be screen. *AUST – NG – J Obst – Gynaecol* 1995 Nov. 35, (4),387-9.
- 38) Pyke DA. Parity and incidence of Diabetes. *Lancet* 1, 818, 1956.
- 39) Serirat et al. Gestational Diabetes *Journal Med. Association Thai* 1992, June 75 (6), 315-9.
- 40) Cousins I. M. Pregnancy compliations among diabetic women. *Rev. 1965-1985. Obst and Gynaecol. Survey* 42-140, 1987.
- 41) Suhoven and Terano et al. Hypertension in preeclampsia in women with gestaional glucose intolerance. *Acta Obst. And gynaecol scand* 2 : 269. 1993.
- 42) Combs CA. Rosenn B, Kitz Miller J. et al. Early prgancy proteinuria in diabetes related to preeclampsia. *Obst and Gynaecol.* 82-801. 1993.
- 43) Rosenn B, Combs CA et al. Poor glycemic control and antepartum obstetric complications in women with insulin dependent diabetes mellitus. *Int. J. Gynaec. Obstet* 43-21, 1993.
- 44) Rodgers BD. Rodgera DE : Clinical variables associated with diabetic ketoacidosis during pregnancy. *J. Reprod. Med.* 36. 797, 1991.

- 45) Jacobson JD. Cousins I : A population based study of maternal and perinatal outcome in patients with gestational diabetes. Am J. Obst. & Gynaecol. 161 – 981, 1989.
- 46) Molsted Pedersen I. Premature labour & perinatal mortality in diabetic pregnancy. Obst. ConsiderationsP. 392. In carbohydrate metabolism in pregnancy and newborn. In Sutherland HW Stowers JM. (eds) New York 1979.
- 47) Phillipson EH. Kallhan SC. Edenberg SC. Williams TG. Maternal obesity at a risk factor to gestational diabetes. Am. J. Perinatal 2 – 268, 1985.
- 48) Goldman et al. Obstet. Complications with GDM. Diabetes – Clinical, Suppl. 2, 40 : 79, 1991.
- 49) Hawthorne G, Robson et al. Prospective population based survey of outcome of pregnancy in diabetic women. Br. Med. J. 1997; 315. 279 – 81.
- 50) Spellacy, WN. Miller, S.Winegeret al. Macrosomia. Maternal characteristic and infant complications. Obstet Gynaecol 1985; 66 : 185.
- 51) Langer C . Is normo glycemia the correct threshold to prevent complications in the pregnant diabetic patient. Diabetes. Rev. 1996; 4 : 2-10.
- 52) High risk pregnancy management options by D.K. James. THIRD EDITION

- 53) Cowett RM. Hypoglycemia and hyperglycemia in the newborn. In : Polin RA, Fox WW, eds. Fetal and neonatal physiology, Philadelphia: W B Saunders, 1997 ; 406.
- 54) Marshall R.E. Infant of diabetic mother; A neonatologists View. Clin. Diabetes, 1990; 8; 49-57.
- 55) Gregory R Scott AR. Mahajer M, Tatter Sal RB. Diabtic pregnancy 1977 – 1990. Have we reached a plateau J coll Physicians London. 26. 162 – 1992.
- 56) O’ Sullivan JB, Mahan CM : Criteria for the oral glucose tolerance test in pregnancy. Diabetes 13 : 278, 1964.
- 57) Anonymous National Diabetic Data Group. Classification and Diagnosis of diabetes mellitus and other categories of gucose intolerance. Diabetes 28 : 1039, 1979.
- 58) Henry EJ : Clinical chemistry : Priciples and techniques I ed Hocher Medical. New York 1965.
- 59) Frienkel N. Banting lecture 1980 of pregnancy. Pregnancy diabetes 19, 1023, 1980.
- 60) Tyrala et al. Infant of diabetic mother Obstet. Gynaecol Clin. North Am. 1996; 23: 221-241.
- 61) Robert MF, Neff RK, Hubbell JT. et al. Association between maternal diabetes and respiratory distress syndrome in the new born. N Eng J Med
- 62) Varley’s clinical biochemistry.

- 63) PROF.V.Seshiah-PRIMER ON DIABETES IN PREGNANCY
- 64) V.Seshiah, V.Balaji,Madhuri.S.Balaji –Rationale For GESTATIONAL DIABETES Screening in pregnancy.

PROFORMA

Serial Number :

Name of the Patient:

Husband's Name :

Age :

OP Number :

Address :

Socioeconomic Status :

Educational Qualification of mother :

OBSTETRIC FORMULA

	Gravida	Parity	Living	Abortion

LMP :

EDD :

Marital Status :

FAMILY HISTORY

Diabetes : Yes / No

Hypertension : Yes / No

GESTATIONAL AGE IN WEEKS AT THE TIME OF TESTING

H/O RISK FACTORS :

IN THE PRESENT PREGNANCY :

Age more than 25 years	
Obesity	
H/o. Recurrent UTI	
History of Moniliasis	
PIH	
Hydramnios	

Glycosuria	
Congenital malformations	

IN THE PAST PREGNANCIES

H/o. Spontaneous Abortions	
H/o. unexplained neonatal death	
H/o. sudden IUD	
H/o. Unexplained still birth	
H/o. Macrosomia	
H/o. GDM	
H/o. PIH	
H/o. Congenital anomalies	

GENERAL EXAMINATION

Anaemia : Yes / No

Pedal Oedema : Yes / No

Pulse Rate :

Blood Pressure :

Height : Weight :

Pre-pregnancy weight :

BMI (Calculated from prepregnancy weight)

CLINICAL EXAMINATION

Gestational Age

Investigations

Urine : Albumin : Sugar :

Blood

Haemoglobin

PCV

DATE OF SCREENING			
Gestational Age at screening			
2 hr 75g WHO OGCT			
2 hr 75g WHO OGTT			

USG FINDINGS**OUT COME OF PRESENT PREGNANCY**

Mode of delivery		
Date of delivery		
Gestational Age At Delivery		
Birth Weight		
Apgar score	:	

NEONATAL COMPLICATIONS

Hypoglycemia		
Hypocalcemia		
Hyperbilirubinemia		
Congenital Anomaly		
RDS		

ABBREVIATIONS

ADA	:	American Diabetes Association
AGT	:	Abnormal Glucose Tolerance
BMI	:	Body Mass Index
BW	:	Birth Weight
DM	:	Diabetes Mellitus
EDTA	:	Ethylene Diamine Tetra Acetic Acid
FPG	:	Fasting Plasma Glucose
G.A	:	Gestational Age
GDM	:	Gestational Diabetes Mellitus
GOD	:	Glucose Oxidase
GTT	:	Glucose Tolerance Test
HAPO	:	Hyperglycemia Adverse Pregnancy Outcome
Hb	:	Hemoglobin
IGT	:	Impaired Glucose Tolerance
IOG	:	Institute Of Obstetrics And Gynecology
IQ	:	Intelligent Quotient
IUD	:	Intra Uterine Death
NDDG	:	National Diabetes Data Group
NonGDM	:	Non Gestational Diabetes Mellitus
OGCT	:	Oral Glucose Challenge Test
OGTT	:	Oral Glucose Tolerance Test
PCV	:	Packed Cell Volume
PIH	:	Pregnancy Induced Hypertension
POD	:	Peroxidase
PPG	:	Post Plasma Glucose
RDS	:	Respiratory Distress Syndrome
SD	:	Standard Deviation
SE Status	:	Socio Economic Status
UTI	:	Urinary Tract Infection
VSD	:	Ventricular Septal Defect

WHO : World Health Organisation

KEY TO MASTER CHART

Age

17-20 years	-	1
21-25 years	-	2
26-30 years	-	3
31-35 years	-	4
36 - 40 years	-	5

Socio Economic Status

Class I	-	A
Class II	-	B
Class III	-	C
Class IV	-	D
Class V	-	E

BMI

≤ 20	-	A
21– 24	-	B
25 - 29	-	C
≥ 30	-	D

FAMILY HISTORY

Positive	-	+
Negative	-	-

GESTATIONAL AGE AT TESTING

16-20 weeks - 1
21-24 weeks - 2
25-28 weeks - 3
29-32 weeks - 4

OBSTETRICS CODE

G1(Gravida 1) - I
G2 (Gravida 2) - II
G3(Gravida 3) - III
G4 (Gravida 4) - IV
G5 (Gravida 5) - V
G6 (Gravida 6) - VI

RISK FACTORS IN PREVIOUS PREGNANCY

Spontaneous Abortion - SA
Macrosomia - M
Sudden Intrauterine Death - SID
Unexplained Still Birth - USB
Unexplained Neonatal Death - UND
History of GDM - HG
History of PIH - HP
Congenital Anomaly - CA

RISK FACTORS IN PRESENT PREGNANCY -

Obesity - OB
Pregnancy Induced Hypertension - PIH
Hydramnios - H

Glucosuria	-	G
Congenital Anomaly	-	CA
Macrosomia	-	M
Recurrent UTI	-	RU
Moniliasis	-	MON

GESTATIONAL AGE AT DELIVERY

Term	-	T
Preterm	-	PT

BIRTH WEIGHT (Kg)

2 – 2.4	-	A
2.5 – 2.9	-	B
3 – 3.4	-	C
3.5 – 3.9	-	D
≥ 4	-	E

APGAR SCORE

8 and above	-	A
5 – 7	-	B
1 – 4	-	C

NEONATAL COMPLICATIONS

Hypoglycemia	-	HG
Hypocalcemia	-	HC
Hyperbilirubinemia	-	HB
Respiratory Distress Syndrome-	RDS	
CA	-	Congenital Anomaly

S.No.	Name	Age	SE Status	Family History	Obstetric Code	GA at time of Testing	BMI	Risk factors in previous pregnancy	Risk factor in present pregnancy	PLASMA GLUCOSE VALUES	
										WHO OG TT	WHO OGCT
1	Nalini	5	E		III	1	A	SA	RU	63	74
2	Ragini	3	D		I	4	B		M,G,PIH	72	65
3	Ilavarasi	1	E	+	I	3	B			95	96
4	Jayanthi	2	C		II	2	B	PIH	G	102	106
5	Sathya	5	D		III	3	B	PIH		62	65
6	Priya	3	C		I	1	B			86	95
7	Sridevi	1	E		I	1	B		H	95	86
8	Ramya	2	B	+	II	2	D		Ob,PIH	112	114
9	Neelavathi	3	E		I	3	C			65	64
10	Nandhini	3	D		II	1	B			73	76
11	Shayeen	3	C		I	3	D		OB	115	117
12	Kanimozhi	3	E		II	2	B		G,M	96	85
13	Lakshmi	4	E		III	3	C	UND		134	128
14	Radhika	3	C	+	I	3	B		G	96	87
15	Devi	3	E		IV	2	C			68	63
16	Margaret	3	D		II	2	B		PIH	76	74
17	Revathi	3	E		I	1	B			106	107
18	Mumtaj	3	C	+	I	4	A			95	93
19	Logeshwari	3	D		III	2	B	USB	M	116	109
20	Vanitha	2	D		I	3	C			109	116
21	Tulasi	3	D	+	III	1	B			74	76
22	Gajalakshmi	1	E		I	2	B			87	89
23	Anandhi	3	C		II	1	A	PIH	G	96	95
24	Fathima	4	D		III	3	B	SA	PIH	66	65
25	maheshwari	3	E	+	I	3	B			74	76
26	Vijayalakshmi	2	E		I	2	D		OB	106	108
27	Gomathi	3	D		III	1	B		G	94	96
28	Shanthi	2	E		I	4	B		RU	116	108
29	Selvi	3	E		I	3	B			65	67
30	Sripriya	2	D		III	4	B	SID		96	95
31	Jothi	3	E		I	1	C			91	95
32	Rama	3	D	+	II	4	D	PIH	OB	116	106
33	Nabeesa	2	C		I	2	B			95	98
34	Raji	3	E		I	3	B			76	84
35	Megala	4	C		II	4	B	UND	G	83	72
36	Rajam	1	E		I	2	B			65	67
37	Melisna	3	E		II	1	B			106	116
38	Dhakshayini	5	C	+	II	3	B			105	109
39	Venkatapriya	2	E		I	3	C		PIH	94	97
40	Latha	3	D		I	1	A			74	77
41	Kalaivani	4	E		II	2	B	SID	M	107	109
42	Mohana	3	E		IV	4	B	SA,M		116	118

43	Anitha	3	D		I	4	B			106	105
44	Priya	3	D		I	2	C			116	112
45	Nirmala	1	D		I	1	B			106	105
46	Jeslin	4	E		III	2	B			86	79
47	kala	2	D	+	I	4	B			78	84
48	Sumati	3	C		II	4	D	M	OB	116	118
49	Sadhana	4	D		II	1	A		G	94	92
50	Indira	4	E		I	3	B			106	109
51	Jesintha	1	D		I	2	B			64	68
52	Subha	1	E		I	4	B			86	78
53	Gantha	4	D		II	4	D		OB,PIH	124	118
54	Bindhu	2	C		I	4	B			76	84
55	Amitha	3	E		II	3	B		G	116	124
56	Govindammal	1	C	+	I	2	B			106	94
57	Reema	3	D		II	4	B	USB		94	108
58	Jamila begum	2	C		I	3	B			116	112
59	Farhana	3	E		I	3	B		RU	112	118
60	Amudha	3	D		I	4	D		OB	132	136
61	Lakshmi	2	D		I	4	B		G	106	108
62	Trisha	3	E		I	4	B			84	82
63	Renuka	3	D		III	4	D	SA	OB	124	116
64	Misha	3	E		I	1	B			126	126
65	Gowri	4	D		II	4	B		MON	116	106
66	Nirupama	2	E		I	3	B			104	116
67	Sarala	1	E		I	4	B			96	88
68	Meenakshi	3	D		I	3	B			84	88
69	Rose	3	C		I	4	B		MON	124	109
70	Jayakumari	3	E		I	2	B			106	121
71	Rasiya begum	3	D	+	I	4	C		PIH	122	126
72	Padmini	4	E		II	4	A	PIH	G	84	86
73	Guna	2	D		I	4	C			121	134
74	Jothiswari	3	E	+	I	4	C		G	104	106
75	Banumathi	3	D		II	4	D		OB	134	128
76	Vijaya	1	D		I	2	B		M	124	128
77	Lekha	3	E		III	3	B			84	85
78	Rajakumari	3	E		II	4	B	SID		106	86
79	Vasantha	3	D		IV	4	B			124	126
80	Aysha	2	C		III	3	B	SA		84	94
81	Velankanni	3	E		I	4	C			94	98
82	Shajith bee	1	D		I	2	C			102	101
83	Bharathi	3	E		II	3	D	PIH	OB	124	126
84	Punitha	2	D		I	3	B			84	83
85	Hema	3	E		I	4	C		G,H	124	128
86	sangeetha	3	E		II	2	B	PIH		121	126
87	Nivetha	1	E		I	4	B		MON	107	94
88	Chithra	4	D	+	II	4	B			124	129
89	Rani	1	D	+	I	2	A		PIH	68	72
90	Latha	3	C		II	1	D	SID	OB,PIH	94	91
91	Hema	2	E		I	3	B		G	121	127
92	Meena	1	E		II	4	C	USB		72	61
93	Saradha	4	D	+	I	2	B		MON	91	95
94	Mari	3	E		I	3	D		OB	124	125

95	Rama	4	E		III	1	B	M	RU	72	62
96	Vijayalakshmi	2	E		I	2	B			124	126
97	Elavarasi	3	D		II	4	C	UNB		62	72
98	Kamini	3	E		I	4	B			106	124
99	Koteeswari	1	E		II	3	B	HG	G	126	121
100	Eswari	3	C	+	I	2	D		OB	116	121
101	Lakshmi	4	D		I	1	A			94	96
102	Nithya	2	E		I	3	B			61	63
103	Jothilakshmi	3	E		II	2	C	M		71	74
104	Sandhya	1	E		I	3	B			94	97
105	Muneeswari	3	D		III	2	B	SA		61	63
106	Mahalakshmi	3	E		I	4	B			72	61
107	Vidhya	1	C		II	1	D		OB	106	108
108	Fousia	2	D		I	2	B			61	71
109	Parveen	3	E		III	3	A	SA		94	104
110	Elakiya	4	E	+	I	1	C		G	112	122
111	Dharini	2	D		I	2	B			104	96
112	Dhanalakshmi	3	E		II	4	D	M	OB,PIH	124	112
113	Iniya	1	C		I	4	B			62	74
114	Ellammal	3	E	+	I	1	B			112	124
115	Rojarani	3	D		II	2	B	SID	G	67	72
116	Ramani	2	E		I	3	C			74	68
117	Rosi	5	E		I	4	D		OB	84	92
118	Ambika	1	D		II	4	B	USB		105	101
119	Veena	3	C		I	2	B			99	100
120	Menaka	4	E		III	1	B	SA		80	78
121	Banu	2	D		I	3	B			66	67
122	Chamundeeswari	3	E		I	4	B			112	124
123	Bindu	3	E		I	3	B			112	104
124	Sivasankari	1	E		II	3	B	PIH		65	76
125	Monisha	2	D		I	1	C		G	96	94
126	Annalakshmi	4	E		IV	2	D	SA	OB	102	112
127	Bragadi	5	C		I	4	B		MON	72	68
128	Vishalam	2	E	+	II	2	B			69	60
129	Durgalakshmi	1	D	+	I	1	A		RU	84	105
130	Malliga	3	E		II	4	C	PCA		107	88
131	Cicillia	3	D		I	3	B		PIH	124	127
132	Tharangini	2	E		I	2	D		OB	122	122
133	Nagammal	3	D		II	1	B	UND	H	67	66
134	Chandra	1	C		I	4	C		G	129	128
135	Poongodi	3	E		II	3	B			94	96
136	Dahlia	2	D		I	2	B			88	90
137	Sumathi	1	E		I	4	C			104	100
138	Ayyammal	3	E		I	3	B			116	110
139	Mala	3	D		III	4	B	SA		96	112
140	kalaiselvi	3	E		II	3	B			106	96
141	Sendamarai	4	D		I	1	B			74	74
142	Shanthi	2	C		II	2	B	PIH	G	94	101
143	Anandhi	1	E		I	3	D		OB	68	74
144	Durga	3	E		I	2	A		PIH	116	118
145	Thenmozhi	2	D		II	1	C		MON	84	86
146	Nagabooshanam	4	E	+	I	3	B			66	69

147	Chellammal	3	E		I	2	B		H	96	106
148	Duraiammal	5	D	+	I	4	D		OB	124	127
149	Ranjani	1	E		III	4	B	SA	PIH	106	100
150	Neelavathi	4	C		I	1	C		G	116	124
151	Chellammal	2	E		II	2	B	PIH		96	94
152	Palaniammal	3	E	+	I	3	D		OB	62	67
153	Deivanai	3	D	+	IV	2	B	SA		126	106
154	Dhanakodi	3	E		I	3	B			74	72
155	Mumtaz	3	E		I	2	B			104	106
156	Loganayaki	2	D		I	4	B			106	116
157	Rajeshwari	3	E		I	2	B		CA	62	64
158	Pounu	1	D		II	1	B			106	88
159	Mayil	2	C		I	3	C			94	104
160	Pattammal	3	E		I	3	C			86	92
161	Kanniammal	4	E		II	2	A			61	64
162	Vijaya	2	D		I	4	D		OB,PIH	126	116
163	Cauvery	1	E	+	III	2	B	SID	G	94	96
164	Poovathal	3	E		I	1	B		H	74	76
165	Yamuna	5	C		II	3	B			112	124
166	Sarojini	3	E		I	2	C			94	92
167	Ammu	1	D		I	4	B			62	67
168	Lalitha	3	E		II	4	A	USB	G	96	106
169	Gowri	2	E		I	2	D		OB	104	98
170	Manohari	1	C		I	3	B			109	108
171	Dhanam	3	D		III	2	C		CA	112	116
172	Amulu	4	E		I	4	B			94	96
173	Kuppu	2	E		II	2	D		OB	124	127
174	Glory	3	E		I	3	B			68	70
175	Kasiammal	2	E		II	4	D		OB	94	87
176	Meenammal	1	E		I	3	B			86	92
177	Nowshath	3	C	+	I	3	D		OB	116	106
178	Anjalai	4	D		II	2	A	PIH	G	62	71
179	Martha	3	E		I	3	C		MON	105	112
180	Ranjitha	1	E	+	III	4	B	SA	RU	94	96
181	Kotteeswari	2	E		I	1	B			74	76
182	Gracy	3	D		I	2	D		OB	81	85
183	Vanaja	4	E		II	3	C	UND	PIH	112	122
184	Samudhram	2	E	+	I	4	B		G	68	67
185	Alisha	3	C		II	1	B	PIH		124	112
186	Yesodha	1	E		I	3	D		OB	94	97
187	Sulochana	4	E		IV	4	C	M,SA		86	8
188	Gunasundari	3	D		I	3	B		CA	124	127
189	Shanthakumari	5	E		I	4	B			94	101
190	Shalini	2	D		I	3	B			106	98
191	Logeshwari	3	D		I	4	C			110	114
192	Boomadevi	3	E		II	3	B	SID		112	109
193	Adhilakshmi	1	D		I	2	B		G	84	88
194	Kanika	2	C		I	3	D		OB	94	96
195	Tamilselvi	3	E	+	II	1	C	HG		104	107
196	Devi	4	E		I	4	A			67	69
197	Geetha	3	D	+	I	2	B		H	124	116
198	Shankari	1	E		I	3	D		OB	112	124

199	Chennammal	4	E		III	1	B	SA		98	90
200	Kanchana	3	E		I	4	B			89	97
201	Amsa	2	D		II	2	C	PCA	PIH	79	80
202	Esthermary	3	C	+	I	3	D		OB	124	127
203	Veni	1	E		I	4	B			86	87
204	Priya	3	D		II	2	B	USB		124	122
205	Malar	4	E		I	3	B			106	98
206	Godavari	2	D		I	4	B			88	8
207	Velankanni	3	E		I	3	B			92	101
208	Ruthra	2	E		I	3	B			81	91
209	Chinnapappa	3	D		II	2	B	PIH	PIH	106	107
210	Umarani	1	E		I	4	B			94	88
211	Mani	2	E		I	3	B			61	64
212	Devammal	3	C		III	1	A	M	PIH	121	127
213	Pamila	4	E	+	I	2	C		MON	86	81
214	Ganga	2	D		I	3	D		OB	110	109
215	Victoria	3	E	+	II	4	B		RU	94	97
216	Rebecca	1	D		I	1	B		G	105	112
217	Kanmani	4	E		II	2	A		H	124	127
218	Sita	3	D		I	3	C			66	69
219	Fathima	2	C	+	IV	4	B			84	89
220	Jeyamani	3	E		I	3	D		OB	122	116
221	Meenalochini	1	D		I	2	B			76	74
222	Pappa	3	E		III	4	B			120	128
223	Sivagami	3	D		I	4	B		G	94	106
224	Alamelu	3	E		I	3	B			108	100
225	Kamala	2	E		I	2	A			98	105
226	Selvarani	3	E		I	3	C		G	90	78
227	Mangai	3	D		II	4	D	M	OB	106	95
228	Jagadha	2	E		I	3	B			68	81
229	Kalyanam	3	C		III	1	B	PIH		80	70
230	Jaya	4	E		I	4	D		OB	129	120
231	Prema	1	D		IV	4	B	SA		100	99
232	Ponni	2	E		I	1	C		PIH,G	110	128
233	Komalavalli	3	D		IV	2	B			120	110
234	Venda	3	D	+	I	3	D		OB	66	65
235	Kannagi	2	C		II	4	B	USB	H	120	116
236	Uma	3	E		I	1	B		PIH	84	87
237	Kanaga	1	D	+	I	2	C			110	98
238	Veda	3	E		II	3	D	SID	OB	100	106
239	Neela	4	D		I	4	B			67	69
240	Rani	2	E		I	3	B			84	87
241	Karpagam	3	D		III	4	D		OB	124	128
242	Kamala	2	E		I	2	B			116	100
243	Radha	3	D		I	3	C			62	67
244	Parvathi	1	E		II	2	B	PIH	MON	100	106
245	Jeba	3	D		I	4	D		OB	90	94
246	Chinnathayi	3	C		III	3	B	M		96	88
247	Rajathi	4	E		I	1	B		PIH	61	71
248	Umapriya	2	D		I	4	C			116	118
249	Sarojini	3	E	+	II	4	B	PIH	PIH	124	126
250	Ramayee	1	D		I	2	B			80	79

251	Baby	3	E	+	I	1	B			86	94
252	Alli	3	C		II	3	B	USB		96	88
253	Therasa	2	D		I	4	C		G	66	64
254	Vanaja	3	E		I	3	B			106	124
255	Ajitha	1	D	+	I	2	B		CA	126	109
256	Salma	4	E		II	4	B			100	98
257	Saraswathy	3	C		II	3	B			89	82
258	Vishalam	4	D		II	4	A		PIH	120	118
259	Anjali	2	E		I	2	A			110	100
260	Prabha	3	E		II	3	C		G	80	77
261	Samitha	2	D		I	4	B		PIH	84	87
262	Selvi	3	E		II	3	D		OB	94	110
263	Amala	4	D		I	1	B		CA	126	128
264	Riswana	1	C	+	I	2	C			94	100
265	Aruna	3	D	+	III	4	B	SA	PIH	121	128
266	Krishnaveni	2	E		I	3	A			90	80
267	Maya	3	D		IV	1	D			78	89
268	Rupa	3	E		I	4	C		OB	130	124
269	Annam	3	D	+	I	2	B			106	104
270	Periyanayaki	1	C		II	3	B	USB		100	98
271	Panchalai	4	D		I	4	B		PIH	130	121
272	Mangammal	3	E		II	3	B			110	116
273	Pramila	2	D		I	2	B			118	104
274	Kavitha	3	E		III	4	B			94	96
275	Kalpana	3	D		I	3	B			116	118
276	Punitha	2	E		I	2	B			61	67
277	Kumudha	1	D		II	3	C			100	89
278	Shanthakumari	3	E		I	4	B			68	70
279	Tamilarasi	3	D		II	4	B	M	MON	90	100
280	Chellammal	4	C	+	I	1	B		PIH	124	110
281	Raji	2	E	+	III	2	C		RU	107	121
282	Ranganayaki	3	D		I	3	D		OB	100	92
283	Kaliyammal	1	E		I	1	A		H	90	96
284	Vaidegi	3	D		II	4	C	SID	G	116	118
285	Manju	4	E		I	2	B			64	71
286	Savithri	2	C		I	3	B		PIH	74	66
287	Maria	3	D	+	III	4	B	SA		106	108
288	Rajini	3	E		I	2	D		OB	90	96
289	Mehaboob	4	D		IV	4	B			95	88
290	Nalini	3	E		I	3	B			80	78
291	Kowsalya	1	D		II	4	B			116	120
292	Chinnaponnu	3	E		I	2	B		PIH	96	98
293	Solay	4	E		I	2	B			94	93
294	Panchalai	2	D		II	3	D		OB,PIH,G	112	116
295	Jayalakshmio	3	E		I	4	A			66	74
296	Ruckmani	3	D		I	2	C		MON	76	61
297	Divya	2	E		II	3	B			62	64
298	Kanaga	3	E	+	I	1	D		OB,H	94	88
299	Pechi	1	C	+	I	3	B	PCA		89	96
300	Pattu	4	D		II	4	B		RU,PIH	124	126
301	Narayani	3	C		I	1	C			74	76
302	Kasthuri	3	E		I	2	B			104	107

303	Pushpa	2	D	+	III	3	B	UND	PIH	124	116
304	Malleswari	4	E		I	4	C		G	118	128
305	Usha	3	D		II	1	D		OB	96	94
306	Kasimbee	1	E		I	2	B			65	64
307	Bharani	3	D		II	3	B			88	87
308	Rajeswari	2	E		I	2	B			94	106
309	Sayeedha	3	D		II	4	D		OB	107	94
310	Chithra	3	E		I	1	B			92	109
311	Maheshwari	1	D		III	2	B	SA		102	91
312	Sulekha	3	E		I	3	C			84	87
313	Ponnammal	2	D		II	2	D		OB	74	77
314	Kamala	3	E		I	3	B			76	72
315	Govindammal	4	D	+	I	1	B			94	97
316	Lakshmi	2	C		II	4	C	PIH	MON	124	127
317	Sundari	3	D		I	2	A			106	104
318	Kalaiarasi	1	E		I	3	D		OB,RU	74	77
319	Vaidegi	4	D	+	I	4	B			94	97
320	Aachi	3	E		I	2	C		G	112	127
321	Santhanalakshmi	2	D		II	3	B	USB		84	87
322	Kamala	3	E		I	4	B			124	118
323	Chithra	3	D		II	3	D	HG	OB	94	97
324	Veeralakshmi	3	E		I	2	C			106	107
325	Selvi	1	D		I	3	B			84	87
326	Muniammal	3	E		IV	3	B	SID		116	118
327	Vanitha	3	E	+	I	1	B		M	94	96
328	Jeynabee	2	E		II	2	D		OB	124	128
329	Surekha	3	C		I	3	B			62	61
330	Kumari	1	D		I	4	B			84	87
331	Pushpa	4	E	+	II	2	C	UND		94	104
332	Dhana	3	E		I	3	B			106	98
333	Violet	2	D		I	4	D		OB	124	132
334	Noor	3	E		II	3	B	USB	PIH	112	114
335	Saraswathy	4	C		I	4	B			66	65
336	Rani	3	D		III	2	C	M	G	84	87
337	Rohini	4	E	+	I	1	D		OB	106	99
338	Revathy	1	D		IV	3	B	SA		94	104
339	Ponni	3	E		I	4	A			72	71
340	Porkodi	2	D		IV	2	B			112	117
341	Lakshmi	3	E		I	3	C			69	70
342	Maragatham	3	D	+	I	4	B		M	84	87
343	Rathi	3	C		II	3	D		OB	104	107
344	Rosy	3	d		I	1	A		M	106	110
345	Jaya	2	E		II	2	B			90	88
346	Lily	1	B		I	3	C		G	69	64
347	Nagalakshmi	4	E	+	I	4	D		OB	96	95
348	Jyothi	3	D		II	2	B	M	M	104	116
349	Shanmugavalli	2	C		I	3	B		PIH	112	116
350	Akila	3	E		I	2	C			66	69
351	Gomathy	3	D	+	II	4	D		OB	88	89
352	Sundhari	4	E		I	2	B			74	72
353	Sakthi	1	D		I	3	B			106	110
354	Sona	2	C		II	4	B		PIH	124	127

355	Jamuna	3	E		I	4	D		OB	104	114
356	Kamakshi	3	D	+	III	3	C	SA	M	116	120
357	Vinodha	2	E		I	2	A			68	69
358	Ramani	3	D		II	4	B			84	89
359	Loganayagi	3	D		I	4	B			101	104
360	Yamini	1	D		IV	4	B		M	107	109
361	Senbagam	3	D		I	1	C			84	87
362	Vimala	2	E		I	4	A			62	67
363	Yamini	3	D		II	2	D		OB	94	98
364	Idhaya	1	C		I	3	B		M	124	128
365	Roshini	3	D	+	II	4	B	PIH	PIH	89	88
366	Jaya	2	E		I	2	B			64	62
367	Vinitha	4	D		II	3	B		G	120	116
368	Buela	3	C		I	4	D		OB	124	132
369	Sandhya	3	C	+	II	1	B	USB		94	106
370	Carolene	5	E		I	4	C			80	78
371	Jaba	3	D		III	2	B		PIH	106	98
372	Krishnaveni	4	E		I	3	D		OB	124	127
373	Vennila	1	D		I	4	B			66	64
374	Valli	3	E		III	2	B	SID	M	112	114
375	Shobana	2	E		I	4	B			82	89
376	Sumitha	3	D		I	3	B		PIH	101	104
377	Shobana	3	E		II	4	C			124	127
378	Selvi	3	D		I	1	B			84	87
379	Devi	2	E		II	4	D		OB	105	107
380	Ponni	1	E		I	2	B		M	124	127
381	Angammal	3	D		I	3	C			100	98
382	Ayyammal	3	E		II	4	B	USB	CA	132	134
383	Ramya	3	D	+	I	2	B		H	126	130
384	Roja	2	C		II	1	D		OB	94	97
385	Malliga	4	D		I	4	A		G	124	126
386	Buela	3	E		III	3	C	SA	RU	116	118
387	Vanitha	1	E	+	I	4	C			80	78
388	Sree	4	E	+	II	2	B		PIH	110	106
389	Kalyani	2	D		I	4	B			90	88
390	Eswari	3	C		IV	3	B	SA		91	94
391	Sujatha	3	E		I	4	B		M	116	118
392	Sumathi	1	D		II	1	B			84	87
393	Prema	3	E	+	I	4	B		G	134	137
394	Dhineshi	2	D		III	4	B			106	105
395	Sridevi	3	E		I	4	B			101	109
396	Sangeetha	2	E		II	2	A		M	112	116
397	Muniammal	3	D		I	2	B			90	94
398	Janani	1	E		I	3	B			97	88
399	Janaki	3	C		II	4	B		G	124	127
400	Kani	3	E		I	2	B			88	85
401	Maha	2	D		I	4	C		CA	105	109
402	Shanathamani	3	E		II	1	D		OB	116	120
403	Begam	3	E	+	I	3	B		G	96	100
404	Ganga	3	D		III	2	B	SA	RU	74	77
405	Shakunthala	2	C		I	4	B			134	136
406	Pushpa	1	E	+	III	4	B	SID		106	108

407	Kalaiarasi	3	D		I	2	B		M	94	96
408	Sundharambal	3	E		I	1	B		CA	84	87
409	Eswari	2	E		IV	3	B			74	77
410	Deivanai	3	D		I	2	B			124	128
411	Devammal	3	C		II	4	B		M	106	108
412	Parvathy	3	E		I	1	B			98	88
413	Vidhyavathy	2	B		II	4	C	PIH	M	104	107
414	Emelda	3	E		I	2	B		G	90	94
415	Ramasubamma	3	C		III	3	A	SA		105	95
416	Yeesamal	1	D		I	4	B		RU	124	127
417	Parameshwari	3	E		II	3	B			76	79
418	Meenammal	4	D		I	2	B		MON	94	104
419	Mekala	2	E	+	I	4	C			104	107
420	Thenmozhi	3	E		II	3	D		OB	134	138
421	Gloria	3	D		I	1	B		M	112	114
422	Padmapriya	3	E		II	2	A		PIH	76	79
423	Parvathy	2	D		I	4	B			104	107
424	Anandhavalli	3	C		III	3	C	MG		96	90
425	Pavunu	1	E		I	4	B		M	120	124
426	Kalaivani	3	D		II	2	D		OB	127	116
427	Gowri meena	2	E		I	4	C			88	96
428	Gnanamani	3	D		II	1	B			116	117
429	Asha	3	E	+	I	1	B			86	87
430	Renuga	3	E		II	4	C		M	105	109
431	Hemavathy	2	D		I	2	B			77	80
432	Thayalanam	3	C		II	3	A			94	97
433	Kumari	1	D		I	4	B	SID		116	124
434	Aach kannu	3	B		II	2	B			104	107
435	Bagavathi ammal	3	D		I	3	C			84	87
436	Banupriya	2	D		IIII	4	D		OB	124	116
437	Umapriya	3	E	+	I	2	C		G	106	96
438	Jennifer	3	C		II	1	B		PIH	96	105
439	Nagarani	1	D		I	3	B			74	74
440	Saritha	3	E		III	4	C	MG		106	116
441	Athabiya	3	D		I	2	B			86	86
442	Anbarasi	2	E		I	4	B		M	116	106
443	Jeyaseeli	3	C		II	3	C			95	97
444	Visalam	1	D	+	I	2	B		PIH	116	118
445	Visalakshi	3	E		I	4	B			104	107
446	Killiammal	3	E		I	4	C			86	84
447	Komala	2	D		II	2	B	PIH	M	124	127
448	Thilagavathy	3	E		I	3	B			74	79
449	Agalya	1	D		I	4	C		G	112	106
450	Esthermary	3	C		III	2	B	SA		84	87
451	Kalarani	3	E		I	3	D		OB	94	9
452	Pappathy	2	D		I	1	A		M	107	116
453	Jeevarathinam	1	B		II	4	B		MON	118	119
454	Nagavalli	3	D	+	I	2	C			80	79
455	Nageshwari	3	E		II	3	B	USB		124	119
456	Asirvatham	2	D	+	1	4	D		OB	104	107
457	Mangammal	3	C		II	4	P	UND		97	94
458	Vijayavalli	3	D		I	1	C		G	116	132

459	Kamini	1	E		I	2	B			110	108
460	Kumudha valli	3	D	+	II	3	B		PIH	134	116
461	Denamuthu	3	E		I	4	B			86	84
462	Marychitra	3	D		I	2	B			116	120
463	Kanagapriya	2	C		I	1	C			110	104
464	Rajeswari	3	D		II	4	C			96	101
465	Roopa	1	E		I	3	A			69	67
466	Suguna	3	C		II	2	B		G	76	75
467	Swarna	3	D	+	I	4	B		RU	124	127
468	Sofiya	2	B		III	4	C	SA		106	100
469	Soliammal	3	D	+	I	3	B		M	88	90
470	Thirumaiselvi	3	E		II	4	D		OB	101	110
471	Jothi	1	C		I	4	B			134	137
472	Hazel	3	D		III	1	C	HG		74	77
473	Thangammal	3	E		I	2	B			65	67
474	Udaya	3	D	+	III	4	B	SA	M	112	124
475	Narahini	2	E		I	3	B		PIH	96	98
476	Sundaravalli	3	D		IV	4	B		G	105	107
477	Niranjini	3	E		I	3	B			124	120
478	Neeraja	3	C		III	4	B	PIH		118	116
479	Sundaradevi	2	D		I	1	B			96	94
480	Cheeniymammal	3	D		I	4	B			104	106
481	Kavithanjali	2	E	+	I	1	B			116	124
482	Sarojammal	3	D		II	3	C	USB		74	76
483	Bhavani	1	E		I	2	C		G	124	120
484	Swarnalakshmi	3	C		I	4	B			88	86
485	Tamilarasi	3	D		III	3	A	SA		94	97
486	Tamilselvi	2	B		I	4	B		M	116	112
487	Ponnammal	3	D	+	II	2	D		OB	132	134
488	Subbulakshmi	3	E		I	1	C			101	105
489	Rahmath begam	1	E		II	3	B	M	MON	116	109
490	Farana	3	D		I	4	C			108	117
491	Kasturi bai	3	C		II	3	A		G	84	87
492	Barathikannamma	2	E		I	4	B		PIH	124	134
493	Anuradha	3	E		III	4	B			72	76
494	Rachol	3	D	+	I	3	B			136	126
495	Ellammal	3	E		II	1	B			106	96
496	Sudha	4	D		I	3	B			98	105
497	Sasikala	3	C		I	3	B			110	108
498	Bala	2	D		III	4	B	SA		107	102
499	Mobina	3	E	+	I	1	A			74	79
500	Johnsirani	1	D		I	4	C		G	112	106
501	Saramma	3	B		II	3	B			88	85
502	Udaya	3	C		I	4	C			94	100
503	Leena	2	D		I	2	B		PIH	130	119
504	Moihana	3	E	+	II	3	C			110	106
505	Asiyabee	3	D		I	4	B		M	88	84
506	Srinivasini	1	E		III	1	B			136	130
507	Usha	3	D		I	3	D		OB	120	138
508	Padmapriya	3	C		II	4	B			74	76
509	Angaiyarkani	2	E	+	I	3	B		G	130	124
510	Shanthi	3	D		I	4	C			104	107

511	Abibha	1	E		III	3	B			96	94
512	Syamala	3	C		I	4	B			112	121
513	Zeelanini	3	D		IV	3	B			104	107
514	Hemavathy	3	E		I	4	B		PIH	91	88
515	Malar	2	D		II	1	B	PIH		110	108
516	Yuvarani	1	E	+	I	2	C			64	62
517	Gowthami	3	D		I	3	C		G	76	74
518	Muneshwari	3	B		III	4	B	USB		84	92
519	Devibala	3	C		I	4	A		MON	112	114
520	Julimargaret	2	D		I	3	B			104	107
521	Sasikala	3	E	+	I	4	B			86	82
522	Suchitra	3	D		I	1	C		M	68	66
523	Zaheera begam	1	E		II	2	B		G	104	116
524	Saira Banu	3	D		III	3	D	SA	OB	126	124
525	Deepa	3	C	+	I	4	B			74	76
526	Thenmozhi	2	D		II	3	C			132	134
527	Deivamalar	3	E		I	1	B			92	96
528	Anjalatchi	3	D		II	4	B			112	114
529	Uma	3	D		III	4	B	PIH		104	107
530	Ezhilarasi	2	E		I	3	B			86	85
531	Ramani	2	E		I	1	C			84	82
532	Pramila	1	D		II	4	B			104	102
533	Vanilla	3	B		I	2	B			77	79
534	Inora	2	E		I	3	A			116	106
535	Julia	1	C		II	4	C		G	96	94
536	Thangamani	3	D		I	3	B			104	112
537	Chengammal	3	B	+	II	4	B			102	104
538	Aiysha	2	D		I	1	D		OB, M	132	137
539	Renugadevi	3	D		I	2	B			86	89
540	Ambika	2	E		III	3	C			72	75
541	Mohana	1	D		I	4	B			118	122
542	Navaneshwari	3	E		III	3	B	SID		97	92
543	Vigneshwari	3	C		I	2	B			122	113
544	Ponselvi	2	D		I	4	C			106	109
545	Sharmila	3	E		III	1	B			82	85
546	Shakila	3	D		I	3	B			112	122
547	Vasuki	3	E		IV	4	B		G	126	118
548	Gowri	3	E		I	4	B			83	96
549	Kala	1	E		II	1	C	PIH		102	105
550	Manimegalai	3	D		I	3	B			92	88
551	Sankari	3	E		I	2	C			82	88
552	Chittammal	2	C		I	4	B		G	122	139
553	Anitha	2	D		II	4	C	USB		78	75
554	Karpagam	3	E	+	I	3	A			113	116
555	Ganga	3	D		I	2	B		M	132	123
556	Ponni	1	E		II	1	B	SID	MON	96	102
557	Thulasi	3	D	+	I	4	B			107	93
558	Bindhu	2	E		III	3	B	SA	OB	103	113
559	Indhu	2	D		III	4	B			115	125
560	Varalakshmi	1	C	+	I	4	C			96	91
561	Sakthi	3	D		III	1	B	SA		122	115
562	Gowrimeena	3	E		I	2	B			86	83

563	Hemalatha	2	D		IV	3	B			103	109
564	Indirani	4	E		I	4	B		PIH	126	128
565	Dhanam	3	E		I	4	B			85	84
566	Masthan bee	1	D		II	2	B		PIH	105	99
567	Emily mary	3	E		I	4	B			66	63
568	Suna	3	D		III	3	C	SA	G	94	106
569	Nilavazhagi	2	C		I	3	A		RU	77	68
570	Nistha	3	D		II	2	C	HG		105	115
571	Barathy	1	E		I	1	B		M	85	86
572	Rathi	3	D	+	II	4	B		CA	111	116
573	Anbu	3	E		I	4	B		G	62	75
574	Sahayamary	2	D		I	3	B			105	103
575	Vanitha	3	C		I	4	C		RU	132	113
576	Jeevalakshmi	1	E	+	II	2	B	PIH		76	72
577	Jeeva	3	D		I	3	B			95	99
578	Jagadeshwari	2	E		II	4	B			111	131
579	Amulamma	3	D		I	3	B			88	83
580	Pushpalatha	3	E		III	4	D	PIH	OB	101	103
581	Jayachitra	4	D		I	3	C			123	125
582	Stella	3	E		I	4	B			94	85
583	Gajalakshmi	1	D		III	1	C			64	69
584	Rekha	3	E		I	3	B		G	85	92
585	Princy	2	D		I	4	B			102	112
586	Kamala	2	E	+	I	3	C		RU	76	72
587	Anajali	3	C		III	2	B	SA		111	106
588	Bhavani	3	D		I	4	C			92	96
589	Monika	3	E		II	3	D		OB	63	75
590	Jamila	3	D		I	4	A			109	103
591	Jenitha	3	E	+	II	3	B		PIH	105	102
592	Kanchana	4	D		I	1	B			112	114
593	Selvi	1	E		II	2	B			72	68
594	Chitrangi	3	D		I	3	C		G	132	131
595	Girija	3	E		IV	4	B			96	103
596	Gunaboopathy	3	D		III	3	B			82	88
597	Selviraja	3	E		I	4	B			102	95
598	Sarala	4	D		III	3	B	SA		123	129
599	Jabeena	3	D		I	4	B			99	89
600	Yaswanthi	3	E		II	2	C			73	74
601	Jeyanthi selvam	3	C		I	4	D		OB	85	92
602	Latha kutti	1	D		III	1	A	SA		105	112
603	Baranikumari	2	E		I	4	C		G	112	104
604	Ramapraba	2	D	+	I	3	B			73	75
605	Soorya	3	E		II	4	B		M	131	137
606	Annaiviolet	3	D		I	4	C			111	113
607	Jeneth	3	E		I	2	B			95	82
608	Dalia	4	E		II	1	B			85	92
609	Betsy	2	C		I	3	B			132	135
610	Amalavathy	1	E		I	4	B		G	112	123
611	Abiba	3	D		I	2	B			125	120
612	Jayashree	3	E		III	3	B	SA		99	95
613	Tulasi	3	D		I	4	C			105	103
614	Mallini	2	E		I	4	B			124	126

615	Amsavalli	3	D		III	4	B		G	122	124
616	Ilayarani	4	D		I	4	B			116	106
617	Elavarasi	3	E		II	1	C			84	87
618	Murugeshwari	3	D		I	2	B			106	116
619	Dhanalakshmi	2	C		III	3	C	PIH		94	97
620	Sheeladevi	1	C		I	4	A		RU	64	63
621	Epsy	3	D		II	2	B		M	104	107
622	Samundeeshwari	3	E	+	I	3	C		G	109	102
623	Palayam	4	D		II	1	B	USB		74	76
624	Deivanai	2	D		I	4	B		PIH	124	120
625	Pornima	2	C		III	3	B	SA		96	95
626	Durga Devi	3	E	+	I	2	D		OB.M	116	124
627	Meenaselvi	5	D		II	4	B			64	67
628	Kalavathy	3	E		I	1	B			90	77
629	Kulanthaiammal	4	D		II	4	B	PIH		105	107
630	Sudhaselvaraj	3	C		I	2	C			74	82
631	Vadivammal	4	E		III	3	B			94	97
632	Noori	3	E		II	4	B		G	124	127
633	Manju	3	E		I	4	B			116	124
634	Anju	5	D		I	1	B			94	92
635	Dilsath	3	D		I	2	C		G	80	78
636	Usha	3	E		II	4	B	PIH		126	116
637	Nirmala	2	C		I	3	C			84	87
638	Mary manjula	4	D		I	2	A			110	107
639	Shalini	3	D	+	II	1	B			134	137
640	Pushpavathy	2	C		I	4	B		RU	96	104
641	Sasirekha	3	E		III	3	C	SA		124	112
642	Sophiya	4	E		I	2	B			72	74
643	Girija	2	D		II	4	B	PIH	G	116	124
644	Kavya	4	C		I	1	B			124	127
645	Kanimozhi	3	D		I	4	B			86	94
646	Kamatchi	4	D		II	2	B			96	88
647	Karpagam	3	E		I	4	B			74	86
648	Selvarani	3	D		IV	4	B	PIH		124	132
649	Sripriya	5	E		III	4	C	SA	G	134	128
650	Venkatalakshmi	3	E		I	4	B			116	112
651	Sadana	5	D		II	4	C			94	88
652	Nalayini	3	E		I	1	D			87	92
653	Salimi	4	D		I	2	C			106	116
654	Suniya	2	C		II	4	B	PIH	G	118	109
655	Fathima	3	C	+	I	3	A			100	88
656	Rajitha	3	D		III	2	B	SA		84	96
657	Ramya	4	D		I	1	C			132	124
658	Vimaldevi	3	E		II	4	B	PIH		76	86
659	Lakshmi	3	D		I	3	B	OB		106	104
660	Kanimozhi	5	C		I	2	B			124	132
661	Rajalakshmi	2	E		III	1	B	SA	G	84	92
662	Veera kumari	3	D	+	I	4	C			127	124
663	Meenakumari	4	D		I	3	B			94	89
664	Sowmiya	3	E		II	4	B	PIH		124	132
665	Ujjaini	3	D		I	2	C			104	106
666	Srikala	4	E		III	4	B	SA		132	128

667	Rameshwari	3	C		III	4	B			120	110
668	Tamilselvi	5	D		I	4	B			106	116
669	Ramapraba	3	E		II	4	B		G	99	88
670	Kalavathy	4	D		I	1	B			87	92
671	Jaya Lakshmi	3	C		I	4	C			116	118
672	Pattammal	2	D		I	3	B		G	74	82
673	Kanchana	3	E	+	II	2	B			106	107
674	Mayoori	3	D		II	4	B			84	87
675	Sindhu	4	C		I	1	C			132	126
676	Baranikumari	3	D		II	4	A		G	106	96
677	Kalaierasi	5	E		I	3	B			110	108
678	Anabika	3	E		III	2	B	SA		124	132
679	Arasi	4	E		II	4	B			84	87
680	Betty	3	E		II	1	B			106	108
681	Buvaneshwari	4	D		III	4	B	SA		124	132
682	Rupavathy	3	E		II	4	B		G	136	126
683	Hemaselvaraj	3	E		IV	4	B	SA		122	124
684	Harini	3	E		II	3	C			126	122
685	Jebamary	4	E		III	4	B	SA		134	136
686	Kalyani	3	C		II	3	B			86	84
687	Logammal	5	E		II	4	B		G	124	116
688	Rajathi	3	E		V	3	B	SA, SID		105	107
689	Bairavi	3	C		II	4	B			112	124
690	Mangammal	4	E		II	2	B		RU	106	108
691	Mala	3	E		III	4	B			74	82
692	Brindia	5	C		II	1	C	PIH		116	124
693	Ileena	4	E		IV	4	B			84	88
694	Pongudi	3	D		III	4	B	SA		128	118
695	Iyyammal	3	E		II	4	B		G	106	96
696	Swetha	4	E		III	3	B	SA		134	134
697	Akila	3	C		IV	4	C			104	106
698	kulanthai Teresa	5	D		II	4	B			84	87
699	Maheshwari	3	E		V	4	B		G	136	128
700	Lilly	3	E		II	4	B			128	132
701	Amudha	3	E		II	4	C			121	118
702	Vennila	5	E		III	4	B	SA	G	86	84
703	Renugadevi	3	C		II	4	B			112	124
704	Shanmathi	2	E		V	3	B			106	107
705	Ramya	3	E		II	4	B			67	65
706	Selviravi	3	E		I	3	C			124	127
707	Saritha	3	E		II	4	B		G	104	112
708	Ambiga	5	E		VI	4	B	SA		114	110
709	Priya	2	E		III	3	B			81	85
710	Jesinthamary	3	C		VI	4	C	M, UND		78	82
711	Kala	5	E		III	4	B	SA	G	106	112
712	Shanmuga valli	3	E		II	3	C			112	114
713	Pragadeswari	5	E		II	4	B			84	87

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